DOCUMENT RESUME

ED 324 135 PS 019 125

TITLE	The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.
INSTITUTION	Agency for Toxic Substances and Disease Registry (DHHS/PHS), Atlanta, GA.
PUB DATE	Jul 88
NOTE	603p.
PUB TYPE	Reports - Research/Technical (143)
EDRS PRICE	MF03/PC25 Plus Postage.
DESCRIPTORS	At Risk Persons; *Child Health; Females; Hazardous Materials; *Incidence; Intervention; *Lead Poisoning; *Metabolism; National Surveys; *Pollution; *Pregnancy; Public Health; Regional Characteristics; Research Methodology; Research Needs; Tables (Data); Toxicology; Wastes
IDENTIFIERS	Pollutant Concentrations; *Pollutants; Screening Programs; Soil Restoration

ABSTRACT

This national study on lead poisoning in children is organized in three parts. Part 1 provides an executive summary. Part 2 presents background information, an overview, findings, and conclusions. Part 3, which constitutes the bulk of the report, discusses terms, issues, and findings concerning lead metabolism, its relationship to lead exposure and adverse effects of lead, and adverse health effects of lead in relationship to public health risk and societal well-being. Also discussed are the number of children exposed to lead in the United States; numbers of children exposed to lead according to lead source; numbers of women of child bearing age and pregnant women exposed to lead; low-level lead sources and children's aggregate exposure to lead; methods and alternatives for reducing environmental exposure to lead for young children and related risk groups; environmental releases of lead as evaluated under the Superfund Amendments and Reauthorization Act of 1986; and exposure to lead and toxicity in children and other related groups. Information gaps, research needs, and recommendations are discussed. Appendices provide numerous tables of data, a draft report on lead-contaminated soil cleanup, the final and proposed national priorities list of waste sites with lead as a contaminant, and methodological details of blood-lead prevalence projections from the National Health and Nutrition Examination Survey II data. References number 255. (RH)

****
* Reproductions supplied by EDRS are the best that can be made *
* from the original document. *
****

U.S. DEPARTMENT OF EDUCATION
Office of Educational Research and Improvement
EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)

☒ This document has been reproduced as received from the person or organization originating it.

☐ Minor changes have been made to improve reproduction quality

- Points of view or opinions stated in this document do not necessarily represent official OERI position or policy

ED324135

The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress

"PERMISSION TO REPRODUCE THIS MATERIAL HAS BEEN GRANTED BY
Michael
Greenwell
TO THE EDUCATIONAL RESOURCES INFORMATION CENTER (ERIC)."

PS019125

THE NATURE AND EXTENT OF LEAD POISONING IN CHILDREN IN THE UNITED STATES: A REPORT TO CONGRESS

Agency for Toxic Substances and Disease Registry
Public Health Service
U.S. Department of Health and Human Services

July 1988

DISCLAIMER

Mention of the name of any company or product does not constitute endorsement by the Agency for Toxic Substances and Disease Registry.

PREFACE

Section 118(f) of the Superfund Amendments and Reauthorization Act (SARA) of 1986 (42 U.S.C. 9618(f)) required the Agency for Toxic Substances and Disease Registry (ATSDR) of the U.S. Public Health Service to prepare a study of lead poisoning in children. Congress specifically requested that the following issues be addressed, as stated in SARA Section 118(f):

(f) Study of Lead Poisoning in Children--

(1) The Administrator of the Agency for Toxic Substances and Disease Registry shall, in consultation with the Administrator of the Environmental Protection Agency and other officials as appropriate, not later than March 1, 1987, submit to the Congress, a report on the nature and extent of lead poisoning in children from environmental sources. Such report shall include, at a minimum, the following information--

(A) an estimate of the total number of children, arrayed according to Standard Metropolitan Statistical Area or other appropriate geographic unit, exposed to environmental sources of lead at concentrations sufficient to cause adverse health effects;

(B) an estimate of the total number of children exposed to environmental sources of lead arrayed according to source or source types;

(C) a statement of the long term consequences for public health of unabated exposures to environmental sources of lead and including but not limited to, diminution in intelligence, increases in morbidity and mortality; and

(D) methods and alternatives available for reducing exposures of children to environmental sources of lead.

(2) Such report shall also score and evaluate specific sites at which children are known to be exposed to environmental sources of lead due to releases, utilizing the Hazard Ranking System of the National Priorities List.

(3) The costs of preparing and submitting the report required by this section shall be borne by the Hazardous Substance Superfund established under subchapter A of chapter 98 Internal Revenue Code of 1954.

Since much of the data needed for this report was not readily available in an appropriate format in the peer-reviewed literature, but was developed specifically for this report, a postponement of the original deadline (March 1, 1987) was granted to ATSDR. The report has undergone extensive peer review by external peer reviewers[1] and a Federal ad hoc panel.[2]

The ATSDR appreciates the efforts of the consultant authors and peer reviewers in developing this report; the final document, as a culmination of this process, is the sole responsibility of ATSDR in fulfillment of its Congressional mandate (see Acknowledgments, page xix).

The ATSDR considers this report to be a significant milestone in the public's understanding of the widespread distribution and effects of childhood lead poisoning. As with all reports to Congress, the recommendations within this report must be weighed against other competing priorities.

---

[1] External peer reviewers are listed on pages xx-xxi.

[2] Members of the Federal ad hoc panel are listed on pages xxii-xxiii.

CONTENTS

Page

TABLES xi
FIGURES xvi
AUTHORS AND CONTRIBUTORS xvii
ACKNOWLEDGMENTS xix
NONFEDERAL PEER REVIEWERS xx
FEDERAL AD HOC ADVISORY COMMITTEE xxii

PART 1

EXECUTIVE SUMMARY 1
- A. RESPONSE TO DIRECTIVES OF SECTION 118(f) OF SARA 3
  - 1. Section 118(f)(1)(A) 3
  - 2. Section 118(f)(1)(B) 6
  - 3. Section 118(f)(1)(C) 9
  - 4. Section 118(f)(1)(D) 11
  - 5. Section 118(f)(2) 13
- B. SUMMARY OF REPORT RECOMMENDATIONS 13
- C. ISSUES, DIRECTIONS, AND THE FUTURE OF THE LEAD PROBLEM 15
  - 1. Issues 15
  - 2. Directions and Future of the Lead Problem 16

PART 2

CHAPTER I. REPORT FINDINGS, CONCLUSIONS, AND OVERVIEW I-1
- A. BACKGROUND INFORMATION TO THE REPORT I-2
  - 1. Historical Perspectives and Discussion of Important Issues ... I-2
  - 2. Lead Metabolism and its Relationship to Exposure, Risk Population Identification and Adverse Health Effects ... I-3
  - 3. Adverse Prenatal and Postnatal Effects of Lead in Children: Relationship to Public Health Risk and Their Relative Persistence ... I-6
- B. MAIN REPORT: QUANTITATIVE EXAMINATION OF LEAD EXPOSURE AND TOXICITY RISK IN CHILDREN AND PREGNANT WOMEN AND STRATEGIES FOR ABATEMENT ... I-8
  - 1. Ranking of Lead-Exposed Children by Geographic Area I-10
  - 2. Numbers of Lead-Exposed Children by Lead Source I-15
  - 3. Number of Women of Childbearing Age and Pregnant Women ... I-21
  - 4. The Issue of Low-Level Lead Sources and Aggregate Lead Exposure of Children in the United States ... I-23
  - 5. Lead Exposure Abatement Strategies and Alternatives I-24
    - Lead in Paint I-25
    - Lead in Ambient Air I-27
    - Lead in Dust and Soil I-27
    - Lead in Drinking Water I-28
    - Lead in Food I-29
    - Nutritional Measures in Primary Prevention of Lead Exposure ... I-30

CONTENTS (continued)

Page

Secondary Exposure Prevention Measures I-31
Secondary Nutritional Measures I-34
Extra-Environmental Prevention Measures I-34
6. A Review of Environmental Releases of Lead Under Superfund I-34
7. Information Gaps, Research Needs, and Recommendations .. I-36
Information Gaps I-37
Research Needs I-38
Recommendations I-39
1. Lead in the Environment of Children I-39
2. Lead in the Bodies of Children I-41
C. CONCLUSIONS AND OVERVIEW I-42
1. Lead as a Public Health Issue I-42
2. Lead in the Environment of Children I-43
3. Lead in the Bodies of Children I-45
4. The Extent of Lead Poisoning in Children in the United States I-46
5. The Problem of In Utero Lead Toxicity: The Extent of Fetal Lead Exposure in the United States I-48
6. Removal or Reduction of Lead in the Child's Environment I-49
7. Future Directions of Lead as a Public Health Problem ... I-50

PART 3
CHAPTER II. INTRODUCTION AND DISCUSSION OF TERMS AND ISSUES II-1
A. INTRODUCTION II-1
B. DISCUSSION OF TERMS AND ISSUES II-6
1. Young Children and Other Groups at Greatest Risk for Lead Exposure and Adverse Health Effects II-6
2. Monitoring Lead Exposure in Young Children and Other Risk Populations II-7
3. Environmental Sources of Lead in the United States with Reference to Young Children and Other Risk Groups II-9
4. Adverse Health Effects of Lead in Young Children and Other Risk Groups and Their Role in Health Risk Assessment II-11

CHAPTER III. LEAD METABOLISM AND ITS RELATIONSHIP TO LEAD EXPOSURE AND ADVERSE EFFECTS OF LEAD III-1
A. LEAD ABSORPTION IN HUMAN POPULATIONS III-1
1 Respiratory Absorption of Lead in Human Populations III-1
2. Gastrointestinal (GI) Absorption of Lead in Human Populations III-2
3. Percutaneous Absorption of Lead in Human Populations ... III-4
4. Transplacental Transfer and Fetal Uptake of Lead in Pregnant Women III-4
B. DISTRIBUTION OF ABSORBED LEAD IN THE HUMAN BODY III-5
1. Lead Uptake in Soft Tissue III-6
2. Lead Uptake in Mineralizing Tissue III-7

CONTENTS (continued)

	Page
C. LEAD EXCRETION AND RETENTION IN HUMAN POPULATIONS	III-7
D. METABOLIC INTERACTIONS OF LEAD WITH NUTRIENTS AND OTHER ACTIVE FACTORS IN HUMAN POPULATIONS	III-9
E. LEAD METABOLISM AND SOME KEY ASPECTS OF LEAD EXPOSURE AND TOXICITY	III-10
1. Lead Metabolism and the Nature of Lead Exposure and Toxicity	III-10
2. Lead Metabolism and the Identification of Risk Populations for Lead	III-12
3. Lead Metabolism and Biological Exposure Indicators	III-13
CHAPTER IV. ADVERSE HEALTH EFFECTS OF LEAD: RELATIONSHIP TO PUBLIC HEALTH RISK AND SOCIETAL WELL-BEING	IV-1
A. THE EXPOSURE (DOSE) INDEX IN ASSESSMENT OF THE ADVERSE HEALTH EFFECTS OF LEAD IN HUMAN POPULATIONS	IV-2
B. MAJOR ADVERSE HEALTH EFFECTS OF LEAD IN CHILDREN	IV-3
1. Effects of Lead on Heme Biosynthesis, Erythrocyte Physiology and Function, and Erythropoietic Pyrimidine Metabolism	IV-3
2. Neurotoxic Effects of Lead in Children	IV-7
3. Other Adverse Effects of Lead on the Health of Young Children	IV-17
C. DOSE-EFFECT/DOSE-RESPONSE RELATIONSHIPS FOR PEDIATRIC LEAD EXPOSURE	IV-20
D. PERSISTENCE OF ADVERSE HEALTH EFFECTS FROM LEAD EXPOSURE IN YOUNG CHILDREN	IV-21
CHAPTER V. EXAMINATION OF NUMBERS OF LEAD-EXPOSED CHILDREN BY AREAS OF THE UNITED STATES	V-1
A. ESTIMATED NUMBERS OF LEAD-EXPOSED CHILDREN IN SMSAs BY SELECTED BLOOD LEAD CRITERION VALUES	V-2
1. Estimation Strategies and Methods	V-3
2. Results	V-7
B. NUMBERS OF LEAD-EXPOSED CHILDREN BY COMMUNITY-BASED SCREENING PROGRAMS	V-16
1. Lead Screening Programs	V-16
2. The NHANES II Study	V-31
C. RANKING OF CHILDREN WITH POTENTIAL EXPOSURE TO PAINT LEAD IN HOUSING	V-33
1. Strategies and Methods	V-33
2. Results	V-34
D. SUMMARY AND OVERVIEW	V-44
1. Lead-Exposed Children in SMSAs	V-44
2. Numbers of Lead-Exposed Children Detected by Screening Programs	V-46
3. Comparison of Prevalences Found in NHANES II Updated Prevalences and U.S. Screening Programs	V-47

CONTENTS (continued)

Page

4. Children with Potential Exposure to Lead Paint in the SMSAs V-48
5. Conclusions and Overview V-49

CHAPTER VI. EXAMINATION OF NUMBERS OF LEAD-EXPOSED U.S. CHILDREN BY LEAD SOURCE VI-1
- A. GENERAL ISSUES VI-1
  - 1. The Level of Exposure Risk in Human Populations by Lead Source VI-1
  - 2. Relationships of External to Internal Lead Exposure on a Total Population Basis VI-3
  - 3. Human Behavior and Other Factors in Source-Specific Population Exposures to Lead VI-8
  - 4. Organization of the Chapter VI-9
- B. NUMBERS OF CHILDREN EXPOSED TO LEAD IN PAINT VI-10
  - 1. Estimation Strategies and Methods VI-11
  - 2. Results VI-13
- C. NUMBERS OF CHILDREN EXPOSED TO LEAD FROM LEADED GASOLINE VI-17
  - 1. Estimation Strategies and Methods VI-21
  - 2. Results VI-22
- D. NUMBERS OF CHILDREN EXPOSED TO LEAD FROM STATIONARY EMISSION SOURCES VI-23
  - 1. Estimation Strategies and Methods VI-26
  - 2. Results VI-27
- E. NUMBERS OF CHILDREN EXPOSED TO LEAD IN DUSTS AND SOILS VI-29
  - 1. Estimation Strategies and Methods VI-31
  - 2. Results VI-33
- F. NUMBERS OF CHILDREN EXPOSED TO LEAD IN DRINKING WATER VI-33
  - 1. Estimation Strategies and Methods VI-36
  - 2. Results VI-37
- G. NUMBERS OF CHILDREN EXPOSED TO LEAD IN FOOD VI-44
  - 1. Estimation Strategies and Methods VI-46
  - 2. Results VI-48
- H. SUMMARY AND OVERVIEW VI-48
  - 1. Paint Lead as an Exposure Source VI-49
  - 2. Gasoline Lead as an Exposure Source VI-51
  - 3. Lead from Stationary Sites as an Exposure Source VI-52
  - 4. Lead in Dust and Soils as an Exposure Source VI-52
  - 5. Lead in Drinking Water as an Exposure Source VI-52
  - 6. Lead in Food as an Exposure Source VI-53
  - 7. Ranking of Lead-Exposed Children by Source VI-53

CHAPTER VII. EXAMINATION OF NUMBERS OF LEAD-EXPOSED WOMEN OF CHILDBEARING AGE AND PREGNANT WOMEN VII-1
- A. STRATEGIES AND METHODS VII-1
  - 1. Methods VII-2
  - 2. Results VII-5

CONTENTS (continued)

Page

CHAPTER VIII. THE ISSUE OF LOW-LEVEL LEAD SOURCES AND AGGREGATE LEAD EXPOSURE OF U.S. CHILDREN VIII-1
- A. NATIONAL/REGIONAL SURVEYS OF BLOOD LEAD LEVELS: BASELINE LEVELS AND SOURCE-RELATED CHANGES IN SURVEY BASELINES VIII-2
- B. USE OF SOURCE-SPECIFIC TRACING METHODS VIII-4
- C. THE USE OF SOURCE-BASED DISTRIBUTIONS OF LEAD INTAKE AND SOURCE-BLOOD LEAD RELATIONSHIPS IN ASSESSING AGGREGATE INTAKE AND POPULATION RISKS VIII-5
- D. BIOKINETIC MODELS OF THE IMPACT OF LEAD SOURCES ON BLOOD LEAD VIII-6
- E. SUMMARY VIII-13

CHAPTER IX. METHODS AND ALTERNATIVES FOR REDUCING ENVIRONMENTAL LEAD EXPOSURE FOR YOUNG CHILDREN AND RELATED RISK GROUPS IX-1
- A. PRIMARY PREVENTION MEASURES FOR LEAD EXPOSURE IX-4
  - 1. Primary Prevention Using Environmental Measures IX-4
  - 2. Primary Prevention Using Combined Environmental and Biological Measures IX-18
- B. SECONDARY PREVENTION MEASURES FOR LEAD EXPOSURE IX-20
  - 1. Environmental Lead Control IX-20
  - 2. Environmental/Biological Prevention Measures IX-24
  - 3. Extra-Environmental Prevention Measures IX-24

CHAPTER X. A REVIEW OF ENVIRONMENTAL RELEASES OF LEAD AS EVALUATED UNDER SUPERFUND X-1
- A. NPL SITES -- PROPOSED AND FINAL X-2
  - Exposure of Children to Lead at NPL Sites X-2
- B. URBAN AREA SITE X-4
  - HRS Scoring at the Boston Urban Site X-6
- C. REVISION OF THE HAZARD RANKING SYSTEM X-10
- D. SUMMARY X-11

CHAPTER XI. LEAD EXPOSURE AND TOXICITY IN CHILDREN AND OTHER RELATED GROUPS IN THE UNITED STATES: INFORMATION GAPS, RESEARCH NEEDS, AND REPORT RECOMMENDATIONS XI-1
- A. INFORMATION GAPS XI-1
- B. RESEARCH NEEDS XI-4
- C. RECOMMENDATIONS XI-5
  - 1. Lead in the Environment of Children XI-6
  - 2. Lead in the Bodies of Children XI-8

REFERENCES R-1

APPENDIX A. TABLES OF INDIVIDUAL SMSAs WITH A POPULATION OF OVER ONE MILLION SHOWING NUMBERS OF YOUNG CHILDREN BY THE AGE OF THEIR HOUSING AND FAMILY INCOME A-1

CONTENTS (continued)

Page

APPENDIX B. TABLES OF INDIVIDUAL SMSAs WITH A POPULATION OF LESS THAN ONE MILLION SHOWING NUMBERS OF YOUNG CHILDREN BY THE AGE OF THEIR HOUSING AND FAMILY INCOME B-1

APPENDIX C. TABLES OF INDIVIDUAL AND MERGED SMSAs WITH POPULATIONS OF LESS THAN 500,000 SHOWING NUMBERS OF YOUNG CHILDREN BY THE AGE OF THEIR HOUSING AND FAMILY INCOME C-1

APPENDIX D. SMSAs RANKED BY NUMBER OF YOUNG CHILDREN IN PRE-1950 HOUSING AS OF 1980 .... D-1

APPENDIX E. LEAD-CONTAMINATED SOIL CLEANUP, DRAFT REPORT E-1

APPENDIX F. FINAL AND PROPOSED NATIONAL PRIORITIES LIST WASTE SITES WITH LEAD AS AN IDENTIFIED CONTAMINANT F-1

APPENDIX G. METHODOLOGICAL DETAILS OF BLOOD LEAD PREVALENCE PROJECTIONS FROM NHANES II DATA G-1

12

TABLES

Table — Page

I-1 Relative persistence of adverse health effects in infants and children I-9

I-2 Estimated numbers of children, who are projected to exceed 15 μg/dl Pb-B, by family income and race, in all SMSAs, 1984 .... I-12

I-3 Summaries of estimated numbers of children 6 months to 5 years old in all SMSAs who are projected to exceed selected levels of blood lead, by urban status, 1984 .... I-13

I-4 Cincinnati, Ohio-Kentucky - SMSA census count of children of all races, 6 months to 5 years old, by family income, urban status, and age of housing, 1980 .... I-16

I-5 Categories of estimation methods for children exposed to lead by source I-17

I-6 Estimated number of women of childbearing age and estimated number of pregnant women and projected numbers above four selected Pb-B criterion values, by race and age, in all SMSAs, 1984 .... I-22

I-7 Categorical tabulation of the elements of primary and secondary prevention of lead exposure in children and related risk groups of the United States I-25

III-1 Comparative dietary lead metabolism in infants and adults III-8

IV-1 Effects of lead on the heme biosynthesis pathway in humans IV-5

IV-2 Summary of major findings from prospective studies of early developmental exposure to lead IV-10

IV-3 Lowest observable effect level for effects in children IV-21

IV-4 Relative persistence of adverse health effects in infants and children IV-24

V-1 Projected percentages of children 0.5 to 5 years old estimated to exceed selected Pb-B criterion values by family income, race, and urban status, who live "inside central city" of SMSAS, 1984 .... V-7

TABLES (continued)

Table		Page
V-2	Projected percentages of children 0.5-5 years old estimated to exceed selected Pb-B criterion values by family income, race, and urban status, who live "outside central city" of SMSAs, 1984	V-8
V-3	Projected percentages of children 0.5-5 years old estimated to exceed selected Pb-B criterion values by family income and race who live in small SMSAs, 1984	V-9
V-4	Estimated numbers of children 0.5-5 years old, who are projected to exceed three levels of blood lead by family income and race living inside central cities in SMSAs, 1984	V-10
V-5	Estimated numbers of children 0.5-5 years old, who are projected to exceed three levels of blood lead by family income and race living not inside central cities in SMSAs, 1984	V-11
V-6	Estimated numbers of children 0.5-5 years old, who are projected to exceed three levels of blood lead by family income and race in small SMSAs, 1984	V-12
V-7	Estimated numbers of children 0.5-5 years old who are projected to exceed 15 μg/dl Pb-B, by family income and race, in all SMSAs, 1984	V-13
V-8	Summary of estimated numbers of children 0.5-5 years old in all SMSAs, who are projected to exceed selected levels of blood lead, by urban status, 1984	V-14
V-9	CDC lead screening classification scheme, 1978-1985	V-18
V-10	CDC lead screening classification scheme as of January 1985, using the hematofluorometer	V-19
V-11	CDC lead screening classification scheme as of January 1985, using chemical analysis of EP	V-19
V-12	Results of screening in U.S. childhood lead poisoning projects, FY 1981	V-21
V-13	Lead poisoning screening of children reported by 27 state and local agencies, FY 1983	V-23
V-14	Current (1985-1986) lead screening activities reported by state and local programs to ATSDR	V-24

TABLES (continued)

Table		Page
V-15	Impact of new CDC lead exposure screening guidelines on the number of lead toxicity cases in New York City	V-27
V-16	Temporal variation of lead toxicity cases in selected lead poisoning screening programs, 1973-1985	V-29
V-17	Lead screening statistics for the Chicago Department of Health, 1981 to 1985, by CDC Classification II, III, or IV ..	V-30
V-18	Rate of first hospitalization and associated mean Pb-B in "asymptomatic" children in Newark, NJ, by year of first admission	V-31
V-19	Blood lead levels in U.S. children by race and age, 1976-1980	V-32
V-20	Ranking of 1980 census SMSAs by number of children 0.5-5 years old living in pre-1950 housing, and total number of young children in SMSA	V-35
V-21	Cincinnati, Ohio-KY-SMSA 1980 census count of children of all races 0.5-5 years old by family income, urban status, and age of housing	V-43
VI-1	Categories of estimation methods for children exposed to lead by source	VI-4
VI-2	Summary of Pb-B levels of a relatively homogeneous white population in the United States	VI-8
VI-3	National best estimate and upper bound of numbers of children under 7 years old in lead-based painted U.S. housing by age of units	VI-13
VI-4	Numbers of U.S. children residing in unsound and lead-based painted housing ranked by age and criteria for deterioration	VI-14
VI-5	Regional best estimate of numbers of children in unsound lead-based painted housing by age and numbers of peeling paint units	VI-15
VI-6	Estimated numbers of U.S. children living in unsound, lead-based painted housing above indicated Pb-B criterion values	VI-17
VI-7	Recent consumption of lead in gasoline	VI-18

TABLES (continued)

Table		Page
VI-8	Estimated numbers of U.S. children falling below indicated Pb-B levels as a result of Pb-gasoline phaseout	VI-23
VI-9	Geometric mean Pb-B levels by distance from smelter for children near Idaho smelter	VI-24
VI-10	Extrapolated pediatric population estimates for stationary lead sources: TRC/LIA and EPA approaches	VI-28
VI-11	GCA/OAQPS estimates of total and child populations exposed to stationary sources of lead	VI-28
VI-12	Numbers of children living in housing classified by housing age	VI-38
VI-13	Children potentially at risk for lead exposure by household plumbing, by age	VI-38
VI-14	Estimated numbers of children at greatest risk of exposure to lead in household plumbing	VI-39
VI-15	Persistence of aging housing stock in occupied U.S. housing inventory	VI-39
VI-16	Lead levels in water samples obtained from water fountains/coolers at naval facilities in Maryland	VI-42
VI-17	Percentage of variably collected water samples exceeding 20 μg/l of lead at different pH levels and by age of house	VI-43
VI-18	Daily mean dietary lead intake by percentiles	VI-45
VI-19	Mean daily dietary lead intake in preschoolers classified as total or normalized daily intake	VI-46
VII-1	Estimated percentages of women of childbearing age exceeding selected Pb-B values, their geometric means and standard deviations by race and age, for populations in all SMSAs, 1984	VII-5
VII-2	Estimated number of women of childbearing age and estimated number of pregnant women and projected numbers above four selected Pb-B criterion values, by race and age, in all SMSAs, 1984	VII-6
VIII-1	Estimated contributions of leaded gasoline combustion to blood lead by various pathways	VIII-5

TABLES (continued)

Table Page

VIII-2 Illustrative modeling balance schemes for average lead intake and uptake in 2-year old children under three scenarios VIII-9

VIII-3 Comparison of integrated Pb uptake/biokinetic model predictions to 1983 measurements in East Helena, MT VIII-12

VIII-4 Children's blood lead levels measured in Omaha, NE, 1971-1977 vs. integrated uptake/biokinetic model predictions and including source levels VIII-13

VIII-5 Children's blood lead levels measured in Silver Valley, ID, 1974-1975 vs. integrated uptake/biokinteic model predictions VIII-14

IX-1 Categorical tabulation of the components of primary and secondary prevention of lead exposure in children and related U.S. risk groups IX-2

IX-2 Abatement costs and no. of units for different site categories at a leaded paint threshold of 1.0 mg/cm^2 in public housing IX-8

IX-3 Estimated abatement costs and no. of units for different site categories at a lead paint threshold of 1.0 mg/cm^2 in single-family FHA housing units IX-9

IX-4 Summary of total pre-1940 lead-painted housing versus deleading activity in selected Massachusetts communities for 1982-June 1986 .... IX-10

IX-5 Percentage of lead-soldered cans in all U.S. manufactured food cans from 1979-1985 .... IX-17

IX-6 Age- and sex-dependent diet lead intakes in the United States at two time periods IX-18

X-1 Most common activities associated with lead waste at NPL sites with lead release X-5

FIGURES

Figure Page

I-1 Percent of U.S. Residences Constructed Before 1940 .... I-4

II-1 Pathways of Lead from the Environment to Man and Body Disposition of Lead II-2

IV-1 Effects of Lead (Pb) on Heme Biosynthesis IV-4

IV-2 Multi-Organ Impact of Reductions of Heme Body Pool by Lead IV-6

IV-3 Dose-Response Curves for Elevation of EP as a Function of Blood Lead Level IV-22

VI-1 Illustrative Log-normal Pb-B Distribution Curve VI-7

VIII-1 Parallel Decreases in Blood Lead Values Observed in the NHANES II Study and Amounts of Lead Used in Gasoline in 1976-1980 .... VIII-3

VIII-2 Schematic Model of Lead Metabolism in Infant Baboons with Compartmental Transfer Coefficients VIII-7

VIII-3 Comparison of Distribution of Measured Blood Lead Levels in Children 1-5 Years of Age, Living Within 1 Mile of E. Helena, MT., Lead Smelter vs. Levels Predicted by Uptake/Biokinetic Model VIII-11

AUTHORS AND CONTRIBUTORS

Principal Authors

Paul Mushak, Ph.D.
Consultant and Adjunct Professor
University of North Carolina/Chapel Hill
811 Onslow Street
Durham, NC 27705

Annemarie F. Crocetti, Dr.P.H.
Consultant and Associate Adjunct Professor
New York Medical College
Apt. 11-A
31 Union Square West
New York, NY 10003

Contributors

Michael Bolger, Ph.D.
Center for Food Safety and Applied
Nutrition (HFF-156)
Food and Drug Administration
Washington, DC 20204

Jeanne Briskin
Office of Drinking Water (WH-550)
U.S. Environmental Protection Agency
Washington, DC 20460

Jeffrey Cohen, M.S.P.H.
Office of Air Quality Planning
and Standards (MD-12)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

J. Michael Davis, Ph.D.
Environmental Criteria and
Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Henry Falk, M.D.
Center for Environmental
Health and Injury Control
Centers for Disease Control
Atlanta, GA 30333

Lester D. Grant, Ph.D.
Environmental Criteria and
Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Sandra Lee, Ph.D.
Office of Solid Waste
and Emergency Response (WH-548A)
U.S. Environmental Protection Agency
Washington, DC 20460

Ronnie Levin
Office of Policy Analysis (PM-221)
U.S. Environmental Protection Agency
Washington, DC 20460

David C. Moore, Ph.D.
Innovative Technology and Special
Projects Division - Room 8232
U.S. Department of Housing and Urban Development
Washington, DC 20410-6000

Robert S. Murphy, M.S.P.H.
Division of Health Examination
Survey - Room 2-58
National Center for Health Statistics
Centers for Disease Control
Hyattsville, MD 20782

Joel Schwartz, Ph.D.
Office of Policy Analysis (PM-221)
U.S. Environmental Protection Agency
Washington, DC 20460

Staff of the Division of Vital Statistics
National Center for Health Statistics
Centers for Disease Control
Hyattsville, MD 20782

ACKNOWLEDGMENTS

The Agency for Toxic Substances and Disease Registry (ATSDR) gratefully acknowledges the efforts of the consultant authors, Drs. Paul Mushak and Annemarie F. Crocetti, for their authorship of the interim and final drafts of this report to Congress.

The Agency also acknowledges the generous contributions of Dr. Joel Schwartz, Office of Policy Analysis, U.S. Environmental Protection Agency (EPA), in carrying out the prevalence modeling for segments of this report and Dr. Lester Grant, Dr. J. Michael Davis, and Ms. Vandy Bradow of EPA's Environmental Criteria and Assessment Office (ECAO), Research Triangle Park, N.C., for their contributions. The Agency thanks Dr. Sandra Lee of EPA's Office of Solid Waste and Emergency Response for preparation of Chapter X of the report.

A number of Federal agencies contributed data, data analyses, and data interpretations for the report, and the Agency particularly wishes to thank Jeanne Briskin, Ronnie Levin and Jeff Cohen of EPA, Dr. David Moore of U.S. Department of Housing and Urban Development (HUD), Dr. Michael Bolger of the Food and Drug Administration (FDA), Robert Murphy of the Center for Disease Control's National Center for Health Statistics, and Staff of the Division of Vital Statistics, National Center for Health Statistics.

Thanks also are due to Drs. Henry Falk and James Pirkle of the Center for Environmental Health and Injury Control, Centers for Disease Control (CDC) and Dr. Kathryn Mahaffey, National Institute for Occupational Safety and Health, CDC, (NIOSH), for helping with preparation of the report.

The Agency wishes to thank those Federal and nonFederal peer reviewers of the report who reviewed the various drafts and offered constructive and valuable comments, and also to thank ATSDR staff member Susan Baburich, who directed gathering of the most recent screening information and who also assisted Dr. Frank Mitchell in overseeing the preparation of the earliest drafts.

Thanks and appreciation are extended to those persons at ECAO/EPA, Research Triangle Park, N.C., who worked diligently in the production of the various drafts of the report: Ivra Bunn, Linda Cooper, Chip Duke, Douglas Fennell, Varetta Holman, Allen Hoyt, Carolyn Norris, Deborah Staves, and especially Miriam Gattis and Lorrie Godley.

NONFEDERAL
PEER-REVIEWERS

Carol R. Angle, M.D.
College of Medicine
University of Nebraska
418 South 82nd Street
Omaha, Nebraska 68114

Charles D. Brokopp, D.P.H.
Bureau of Preventive Medicine
Idaho Department of Health
Boise, Idaho 83720

J. Julian Chisolm Jr. M.D.
Johns Hopkins University
School of Medicine and
Kennedy Institute
707 N. Broadway
Baltimore, MD 21205

Jerome Cole, Sc.D.
International Lead-Zinc
Research Organization
2525 Meridian Parkway
P.O. Box 12036
Research Triangle Park, NC 27709

Devra Lee Davis, Ph.D.
Board of Toxicology and Environmental
Health Hazards
National Academy of Sciences
2101 Constitution Ave.
Washington, DC 20418

Claire Ernhart, Ph.D.
Department of Psychiatry
Cleveland Metropolitan General Hospital
Cleveland, OH 44109

Patricia Field, Ph.D.
Toxicology Laboratory
State Laboratory of Hygiene
465 Henry Mall
Madison, Wisconsin 53706

Robert A. Goyer, M.D.
Pathology Department
University of Western Ontario
London N6A5C1
Canada

John W. Graef, M.D.
Lead/Toxicology Clinic
Children's Hospital Medical Center
300 Longwood Avenue
Boston, MA 02115

Philippe Grandjean, M.D., Ph.D.
Department of Environmental Medicine
Odense University
J. B. Winslowsvej 19
5000 Odense, Denmark

Paul Hammond, D.V.M., Ph.D.
University of Cincinnati
Institute of Environmental Health
Kettering Laboratories
3223 Eden Avenue
Cincinnati, OH 45267

Philip Landrigan, M.D., M.S.C.
Environmental Health Sciences
Laboratory
Mt. Sinai School of Medicine
New York, NY 10029

Donald Lynam, Ph.D.
Department of Air Conservation
Ethyl Corporation
451 Florida Blvd.
Baton Rouge, LA 70801

Herbert Needleman, M.D.
Department of Psychiatry
Children's Hospital of Pittsburgh
Pittsburgh, PA 15213

Sergio Piomelli, M.D.
Pediatric Hematology - Oncology
College of Physicians and Surgeons
Columbia University
630 West 168th Street
New York, NY 10032

Routte J. Reigart, M.D.
Department of Pediatrics
Medical University of South Carolina
171 Ashley Street
Charleston, SC 29425

John F. Rosen, M.D.
Division of Pediatric Metabolism
Albert Einstein College of Medicine
Montefiore Medical Center
111 East 210th Street
Bronx, New York, NY 10467

Samuel Schwartz, M.D.
Minneapolis Medical Research
Foundation
501 Park Avenue
Minneapolis, Minnesota 55415

Ellen Silbergeld, Ph.D.
Environmental Defense Fund
1525 18th Street, N.W.
Washington, DC 20036

(Listing of nonfederal peer reviewers does not necessarily denote each reviewer's agreement with all the report's content.)

23

FEDERAL AD HOC ADVISORY COMMITTEE

Michael Bolger, Ph.D.
Center for Food Safety and Applied
Nutrition (HFF-156)
Food and Drug Administration
Washington, DC 20204

Jeanne Briskin
Office of Drinking Water (WH 550)
U.S. Environmental Protection Agency
Washington, DC 20008

Rufus Chaney, Ph.D
U.S. Department of Agriculture
Building 008 BARC - West
Beltsville, Maryland 20705

Jeffrey Cohen, M.S.P.H.
Office of Air Quality Planning
and Standards (MD-12)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

J. Michael Davis, Ph.D.
Environmental Criteria and
Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Henry Falk, M.D.
Center for Environmental Health
and Injury Control
Centers for Disease Control
Atlanta, GA 30333

Lester D. Grant, Ph.D.
Environmental Criteria and
Assessment Office (MD-52)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Vernon N. Houk, M.D.
Center for Environmental Health
and Injury Control
Centers for Disease Control
Atlanta, GA 30333

Barry L. Johnson, Ph.D.
Agency for Toxic Substances and
Disease Registry
Atlanta, GA 30333

Sandra Lee, Ph.D.
Office of Solid Waste and
Emergency Response (WH-548A)
U.S. Environmental Protection Agency
Washington, DC 20460

Ronnie Levin
Office of Policy Analysis
U.S. Environmental Protection Agency
Washington, DC 20460

Jane S. Lin-Fu, M.D.
Division of Maternal and Child
Health - Room 6-17
Health Resources and Services
Administration
Rockville, MD 20857

Kathryn R. Mahaffey, Ph.D.
National Institute of
Occupational Safety and Health
Centers for Disease Control
Cincinnati, OH 45226

David C. Moore, Ph.D.
Innovative Technology and Special
Projects Division - Room 8232
U.S. Department of Housing and
Urban Development
Washington, DC 20410-6000

Robert S. Murphy, M.S.P.H.
Division of Health Examination
Survey - Room 2-58
National Center for Health
Statistics
Centers for Disease Control
Hyattsville, MD 20782

David Otto, Ph.D.
Clinical Studies Division (MD-58)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

James Pirkle, M.D.
Center for Environmental Health
and Injury Control
Centers for Disease Control
Atlanta, GA 30333

Rebecca J. Schilling, D.V.M., M.P.H.
Center for Environmental Health
and Injury Control
Centers for Disease Control
Atlanta, GA 30333

Joel Schwartz, Ph.D.
Office of Policy Analysis (PM 221)
U.S. Environmental Protection Agency
Washington, DC 20460

James Simpson
Center for Environmental Health
and Injury Control
Centers for Disease Control
Atlanta, GA 30333

John Zipeto, P.E., M.P.H.
EPA Region 1
JFK Building
Boston, MA 02115

PART 1

EXECUTIVE SUMMARY

Exposure to lead continues to be a serious public health problem -- particularly for the young child and the fetus. The primary target organ for lead toxicity is the brain or central nervous system, especially during early child development. In children and adults, very severe exposure can cause coma, convulsions, and even death. Less severe exposure of children can produce delayed cognitive development, reduced IQ scores, and impaired hearing -- even at exposure levels once thought to cause no harmful effects. Depending on the amount of lead absorbed, exposure can also cause toxic effects on the kidney, impaired regulation of vitamin D, and diminished synthesis of heme in red blood cells. All of these effects are significant. Furthermore, toxicity can be persistent, and effects on the central nervous system (CNS) may be irreversible.

In recent years, a growing number of investigators have examined the effects of exposure to low levels of lead on young children. The history of research in this field shows a progressive decline in the lowest exposure levels at which adverse health effects can be reliably detected. Thus, despite some progress in reducing the average level of lead exposure in this country, it is increasingly apparent that the scope of the childhood lead poisoning problem has been, and continues to be, much greater than was previously realized.

The "Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress" was prepared by the Agency for Toxic Substances and Disease Registry (ATSDR) in compliance with Section 118(f) of the 1986 Superfund Amendments and Reauthorization Act (SARA) (42 U.S.C. 9618(f)). This Executive Summary is a guide to the structure of the document and, in particular, to the organization of the responses to the specific directives of Section 118(f). It also provides an overview of issues and directions to the U.S. lead problem.

The report comprises three parts: Part 1, consisting of the Executive Summary; Part 2, consisting of Chapter I. "Report Findings, Conclusions, and Overview," which provides a more detailed overview of information and conclusions abstracted from the main body of the report; and Part 3, consisting of Chapters II through XI, which constitute the main body of the report.

Before addressing the specific directives of Section 118(f), it is important to point out that childhood lead poisoning is recognized as a major public health problem. In a 1987 statement, for example, the American Academy of Pediatrics notes that lead poisoning is still a significant toxicological hazard for young children in the United States. It is also a public health problem that is preventable.

In recognition of evolving scientific evidence of the harmful effects of lead exposure, Congress directed ATSDR to examine (1) the long-term health implications of low-level lead exposure in children; (2) the extent of low-level lead intoxication in terms of U.S. geographic areas and sources of lead exposure; and (3) methods and strategies for removing lead from the environment of U.S. children.

The childhood lead poisoning problem encompasses a wide range of exposure levels. The health effects vary at different levels of exposure. At low levels, the effects on children, as stated subsequently in this report, may not be as severe or obvious, but the number of children adversely affected is large. Moreover, as adverse health effects are detected at increasingly lower levels of exposure, the number of children at risk increases. At intermediate exposure levels, the effects are such that a sizable number of U.S. children require medical and other forms of attention, but usually they do not need to be hospitalized, nor do they need conventional medical treatment for lead poisoning. For these children, the only appropriate solution, at present, is to eliminate or reduce all significant sources of lead exposure in their environment. At high levels, the effects are such that children require immediate medical treatment and follow-up. Various clinics and hospitals, particularly in larger cities, continue to report such cases.

Lead exposure may be characterized in terms of either external or internal concentrations. External exposure levels are the concentrations of lead in environmental media such as air or water. For internal exposure, the most widely accepted and commonly used measure is the concentration of lead in blood, conventionally denoted as micrograms of lead per deciliter (100 ml) of whole blood -- abbreviated µg/dl. For example, when ATSDR estimated the number of children considered to be at risk for adverse health effects, the Agency used blood lead (Pb-B) levels of 25, 20, and 15 μg/dl to group children by their degree of exposure.

These levels are not arbitrary. In 1985 the Centers for Disease Control (CDC) identified a Pb-B level of 25 μg/dl along with an elevated erythrocyte protoporphyrin level (EP) as evidence of early toxicity. For a number of practical considerations, CDC selected this level as a cutoff point for medical referral from screening programs, but it did not mean to imply that Pb-B levels below 25 μg/dl are without risk. More recently, the World Health Organization (WHO), in its 1986 draft report on air quality guidelines for the European Economic Community, identified a Pb-B level of 20 μg/dl as the then-current upper acceptable limit. In addition, the Clean Air Scientific Advisiory Committee to the U.S. Environmental Protection Agency (EPA) has concluded that a Pb-B level of 10 to 15 μg/dl in children is associated with the onset of effects that "may be argued as becoming biomedically adverse". In this connection, the available evidence for a potential risk of developmental toxicity from lead exposure of the fetus in pregnant women also points towards a Pb-B level of 10 to 15 μg/dl, and perhaps even lower. These various levels represent an evolving understanding of low-level lead toxicity. They provide a reasonable means of quantifying aspects of the childhood lead poisoning problem as it is currently understood. With further research, however, these levels could decline even further.

A. RESPONSE TO DIRECTIVES OF SECTION 118(f) of SARA

Section 118(f) and its five directives give ATSDR the mandate to prepare this report. These directives are identified in the five subsections below.

1. Section 118(f)(1)(A)

This subsection requires an estimate of the total number of children, arrayed according to Standard Metropolitan Statistical Area (SMSA) or other appropriate geographic unit, who are exposed to environmental sources of lead at concentrations sufficient to cause adverse health effects. Chapter V, "Examination of Numbers of Lead-Exposed Children by Areas of the United States," and Chapter VII, "Examination of Numbers of Lead-Exposed Women of Childbearing Age and Pregnant Women," respond to this directive.

28

Valid estimates of the total number of lead-exposed children according to SMSAs or some other appropriate geographic unit smaller than the Nation as a whole cannot be made, given the available data. The only national data set for Pb-B levels in children comes from the National Health and Nutrition Examination Survey II (NHANES II) of CDC's National Center for Health Statistics. The NHANES II statistical sampling plan, however, does not permit valid estimates to be made for geographic subsets of the total data base.

In this report, the numbers of white and black children (ages 6 months to 5 years) living in all SMSAs are quantified according to selected blood lead levels and 30 socioeconomic and demographic strata. Within large SMSAs (those with over 1 million residents each) for 1984, an estimated 1.5 million children had Pb-B levels above 15 μg/dl. In smaller SMSAs (with fewer than 1 million residents), an estimated 887,000 children had Pb-B levels above 15 μg/dl.

In short, about 2.4 million white and black metropolitan children, or about 17% of such children in U.S. SMSAs, are exposed to environmental sources of lead at concentrations that place them at risk of adverse health effects. This number approaches 3 million black and white children if extended to the entire U.S. child population. If the remaining racial categories are included in these totals, between 3 and 4 million U.S. children may be affected. The numbers of children in SMSAs with blood lead levels above 20 and 25 μg/dl are 715,000 (5.2%) and 200,000 (1.5%), respectively. These figures, however, are for all strata combined; many strata (e.g., black. inner-city, or low-income) have much higher percentages of children with elevated Pb-B levels.

Although these projected figures, based on the NHANES II survey, provide the best estimate that can now be made, they were derived from data collected in 1976-1980 (the years of NHANES II) and extrapolated to 1984. With respect to bounds to the above projections, variables in the methods used to generate these figures contribute to both overestimation and underestimation. The major source of overestimation is the unavoidable omission of declines in food lead that may have occurred in the interval 1978-1964 and that would have affected the results of the projection methodology. On the other hand, two significant factors contribute to underestimation. One is the restriction of the estimates to the SMSA fraction of the U.S. child population, some 75% to 80% of the total population. The other is the unavoidable omission of children of Hispanic, Asian, and other origins in the U.S. population. In a number of SMSAs in the West and South west, children in such segments outnumber black children. In balancing all sources of overestimates and underestimates, including variance in the projection model itself, the projections given are probably close to the actual values.

A breakdown of the above estimates according to national socioeconomic and demographic strata shows that no economic or racial subgrouping of children is exempt from the risk of having Pb-B levels sufficiently high to cause adverse health effects. Indeed, sizable numbers of children from families with incomes above the poverty level have been reported with Pb-B levels above 15 µg/dl. Nevertheless, the prevalence of elevated Pb-B levels in inner-city, underprivileged children remains the highest among the various strata. Although the percentage of children with elevated Pb-B levels is not as high in, for example, the more affluent segment of the U.S. population living outside central cities, the total number of children with these demographic characteristics is much greater than the number of poor, inner-city children. Consequently, the absolute numbers of children with elevated Pb-B levels are roughly equivalent for some of these rather different strata of the U.S. child population.

In this report, ATSDR has also used data from lead screening programs and 1980 U.S. Census data on age of housing to estimate SMSA-specific numbers of children exposed to lead-based paint. In December 1986, ATSDR conducted a survey of lead screening programs. Of 785,285 children screened in 1985, 11,739 (1.5%) had symptoms of lead toxicity by one of two definitions. Because CDC criteria for lead toxicity changed in 1985, some programs were still using the 1978 CDC criteria (Pb-B ≥30 µg/dl and EP ≥50 µg/dl) in 1985, whereas others used the new 1985 CDC criteria (Pb-B ≥25 µg/dl and EP ≥35 µg/dl).

Differences in the estimates of children with lead toxicity become apparent when using the NHANES II data and the childhood lead screening program data. Estimates derived from screening program data very likely underestimate the actual magnitude of childhood lead exposure by a considerable margin. This is especially evident when the percentages of positive test results from screening programs are compared with the much higher NHANES II prevalences of elevated Pb-B levels in strata corresponding to screening program target groups, for example, poor, inner-city children in major metropolitan areas.

An analysis of 318 SMSAs, based on 1980 Census data on age of housing, showed that 35 SMSAs had 50% or more of the children living in housing built before 1950. A total of 4,374,600 children (from these 318 SMSAs alone) lived in pre-1950 housing. The percentage of these children with lead exposures sufficient to cause adverse health effects could not be estimated, but the older housing in which they live is likely to contain paint with the highest levels of lead and is, therefore, likely to pose an elevated risk of dangerous lead exposure. A noteworthy finding concerns the distribution of children in older housing according to family income. Actual enumerations (not estimates) show that children above the poverty level constitute the largest proportion of children who reside in older housing. The implication, consistent with the conclusion based on projections from NHANES II data that was stated above, is that children above the poverty level are not exempt from lead exposure at levels sufficient to place them at risk for adverse health effects. Children above the poverty level are the most numerous group within the U.S. child population.

Although Section 118(f)(1)(A) does not explicitly request such information, an accurate description of the full childhood lead poisoning problem requires an estimate of the number of fetuses exposed to lead *in utero*, given the susceptibility of the fetus to low-level lead-induced disturbances in development that first become evident at birth or even some time later during early childhood. Accordingly, in a given year, an estimated 400,000 fetuses (within SMSAs alone) are exposed to maternal Pb-B levels of more than 10 μg/dl and are therefore at risk for adverse health effects. This number pertains to a single year; the cumulative number of children who have been exposed to undesirable levels of lead during their fetal development is much greater, particularly in view of the higher average levels of exposure that prevailed in past years.

2. Section 118(f)(1)(B)

This subsection requires an estimate of the total number of children exposed to environmental sources of lead arrayed according to source or source types. Chapters VI ("Examination of Numbers of Lead-Exposed Children in the United States by Lead Source") and VIII ("The Issue of Low-Level Lead Sources and Aggregate Lead Exposure of Children in the United States") respond to this directive.

The six major environmental sources of lead are paint, gasoline, stationary sources, dust/soil, food, and water. Dust/soil is more properly classified as a pathway rather than a source of lead, but since it is often referred to as a source, it is included. (Figure 11-1 in the main report shows how lead from these sources reaches children.) The complex and interrelated pathways from these sources to children severely complicate efforts to determine source-specific exposures. Consequently, exact counts of children exposed to specific sources of lead do not exist.

The first step in approximating the number of children exposed to lead from each of the six major sources is to define what constitutes exposure. For each lead source, approximate exposure categories are defined and range from potential exposures through actual exposures known to cause lead toxicity. Because the type and availability of data for each lead source vary considerably, definitions of exposure categories also differ for each lead source. The total numbers of children estimated for each source and category are therefore not comparable and cannot be used to rank the severity of the lead problem by source of exposure in a precise, quantitative way. Furthermore, because of the nature of methods used to calculate the numbers of children in these exposure categories, it is not possible to provide estimate errors. Some numbers are best estimates, but others may represent upper bounds or lower bounds.

One should not overlook the limitations and caveats for these calculations, lest the estimates be misinterpreted and misapplied. In addition, source-based exposure estimates of children have different levels of precision. The estimated number of children potentially exposed to a given lead source at any level is necessarily greater than the number actually exposed at a level sufficient to produce a specified Pb-B value. Source-specific estimates of potentially and actually exposed children, based on the best available information and reasonable assumptions, are summarized as follows:

- For leaded paint, the number of potentially exposed children under 7 years of age in all housing with some lead paint at potentially toxic levels is about 12 million. About 5.9 million children under 6 years of age live in the oldest housing, that is, housing with the highest lead content of paint. For the oldest housing that is also deteriorated, as many as 1.8 to 2.0 million children are at elevated risk for toxic lead exposure.

  The number of young children likely to be exposed to enough paint lead to raise their Pb-B levels above 15 μg/dl is estimated to be about 1.2 million.

- An estimated 5.6 million children under 7 years old are potentially exposed to lead from gasoline at some level.

  Actual exposure of children to lead from gasoline, was projected, for 1987, to affect 1.6 million children up to 13 years of age at Pb-B levels above 15 μg/dl.

- The estimated number of children potentially exposed to U.S. stationary sources (e.g., smelters) is 230,000 children.

  The estimated number of children exposed to lead emissions from primary and secondary smelters sufficient to elevate Pb-B concentrations to toxic levels is about 13,000; estimates for other stationary sources are not available.

- The number of children potentially exposed to lead in dust and soil can only be derived as a range of potential exposures to the primary contributors to lead in dust and soil, namely, paint lead and atmospheric lead fallout. This range is estimated at 5.9 million to 11.7 million children.

  The actual number of children exposed to lead in dust and soil at concentrations adequate to elevate Pb-B levels cannot be estimated with the data now available.

- Because of lead in old residential plumbing, 1.8 million children under 5 years old and 3.0 million children 5 to 13 years old, are potentially exposed to lead; for new residences (less than 2 years old), the corresponding estimates of children are 0.7 and 1.1 million, respectively.

  Some actual exposure to lead occurs for an estimated 3.8 million children whose drinking water lead level has been estimated at greater than 20 µg/l.

  EPA, in a recent study, estimated that 241,000 children under 6 years old have Pb-B levels above 15 µg/dl because of elevated concentrations of lead in drinking water. Of this number, 100 have Pb-B levels above 50 µg/dl, 11,000 have Pb-B levels between 30 and 50 µg/dl, and 230,000 have Pb-B levels between 15 and 30 µg/dl.

- Most children under 6 years of age in the U.S. child population are potentially exposed to lead in food at some level.

  Actual exposure to enough lead in food to raise Pb-B levels to an early toxicity risk level has been estimated to impact as many as 1 million U.S. children.

Despite limitations in the precision of the above estimates, relative judgments can be made about the impact of different exposure sources. Some key findings are:

- As persisting sources for childhood lead exposure in the United States, lead in paint and lead in dust and soil will continue as major problems into the foreseeable future.

- As a significant exposure source, leaded paint is of particular concern since it continues to be the source associated with the severest forms of lead poisoning.

- Lead levels in dust and soil result from past and present inputs from paint and air lead fallout and can contribute to significant elevations in children's body lead burden (i.e., the accumulation of lead in body tissues).

- In large measure, paint and dust/soil lead problems for children are problems of poor housing and poor neighborhoods.

- Lead in drinking water is a significant source of lead exposure in terms of its pervasiveness and relative toxicity risk. Paint and dust and soil lead are probably more intense sources of exposure.

- Greater attention must be paid to lead exposure sources away from the home, especially lead in paint, dust, soil, and drinking water in and around schools, kindergartens, and similar locations.

- The phasing down of lead in gasoline has markedly reduced the number of children impacted by this source as well as the rate at which lead from the atmosphere is deposited in dust and soil.

- Lead in food has been reduced to a significant degree in recent years and contributes less to body burdens in the United States than in the past.

- Significant exposure of unkown numbers of children can also occur under special circumstances: renovation of old houses with lead-painted surfaces, secondary exposure to lead transported home from work places, lead-glazed pottery, certain folk medicines, and a variety of others unusual sources.

3. Section 118(f)(1)(C)

This subsection requires a statement of the long-term consequence for public health of unabated exposure to environmental sources of lead. Chapters III ("Lead Metabolism and Its Relationship to Lead Exposure and Adverse Effects of Lead") and IV ("Adverse Health Effects of Lead") address this issue.

Infants and young children are the subset of the U.S. population considered most at risk for excessive exposure to lead and its associated adverse health effects. In addition, because lead is readily transferred across the placenta, the developing fetus is at risk for lead exposure and toxicity. For this reason, women of childbearing age are also an identifiable, albeit surrogate, subset of the population of concern, not because of direct risk to their health, but because of the vulnerability of the fetus to lead-induced harmful effects.

Direct, significant impacts of lead on target organs and systems are evident across a broad range of exposure levels. These toxic effects may range from subtle to profound. In this report, the primary focus has been on effects that are chronic and that are induced at levels of lead exposure not uncommon in the United States. Cases of severe lead poisoning are, however, still being reported, particularly in clinics in our major cities.

The primary target organ for lead toxicity is the brain or central nervous system (CNS), especially during early child development. Other key targets in children are the body heme-forming system, which is critical to the production of heme and blood, and the vitamin D regulatory system, which involves the kidneys and plays an important role in calcium metabolism. Some of the major health effects of lead and the lowest-observed-effect levels (in terms of Pb-B concentrations) at which they occur can be summarized as follows:

- Very severe lead poisoning with CNS involvement commonly includes coma, convulsions, and profound, irreversible mental retardation and seizures, and even death. Poisoning of this severity occurs in some persons at Pb-B levels as low as 80 μg/dl. Less severe but still serious effects, such as peripheral neuropathy and frank anemia, may start at Pb-B levels between 40 and 80 μg/dl.

- Numerous epidemiologic studies of children have related lower levels of lead exposure to a constellation of impairments in CNS function, including delayed cognitive development, reduced IQ scores, and impaired hearing. For example, peripheral nerve dysfunction (reduced nerve conduction velocities) have been found at Pb-B levels below 40 μg/dl in children. In addition, deficits in IQ scores have been established at Pb-B levels below 25 μg/dl. Preliminary data suggest that effects on one test of children's intelligence may be associated with childhood Pb-8 levels below 10 μg/dl.

- Adverse impacts on the heme biosynthesis pathway and on vitamin D and calcium metabolism, all of which have far-reaching physiological effects, have been documented at Pb-B levels of 15 to 20 μg/dl in children. At levels around 40 μg/dl, the effects on heme synthesis increase in number and severity (e.g., reduced hemoglobin formation).

- Of particular concern are consistent findings from several recent longitudinal cover a period of years epidemiologic studies showing low-level lead effects on fetal and child development, including neurobehavioral and growth deficits.

These effects are associated with prenatal exposure levels of 10 to 15 μg/dl.

With regard to the long-term consequences of lead exposure during early development, the American Academy of Pediatrics (1987) has noted that utmost concern should be given to the irreversible neurological consequences of childhood lead poisoning. Recent findings from longitudinal follow-up studies of infants starting at birth (or even before birth) show persistent deficits in mental and physical development through at least the first two years of life as a function of low-level prenatal lead exposure. It is not yet known, however, whether deficits in later childhood development will continue to show a significant linkage to prenatal exposure or whether, at older ages, postnatal lead levels will overshadow the effects of earlier exposure. Human development is quite plastic, with well known catch-up spurts in growth and other aspects of development. On the other hand, even if early lead-induced deficits are no longer detected at later ages, this apparent recovery does not necessarily imply that earlier impairments are without consequence. In view of the complex interactions that figure into the cognitive, emotional, and social development of children, compensations in one facet of a child's development may exact a cost in another area. Very little information is available for evaluating such interdependencies and trade-offs, but at this point even "temporary" developmental perturbations cannot be viewed as inconsequential.

In addition, given the poor prospects for immediate improvements in the environments of many children (e.g., deteriorated housing occupied by underprivileged, inner-city children), lead exposure and toxicity often are, in practice, irreversible. Thus, the issue of persistence must encompass the reality of exposure circumstances as well as the potential for biological recovery.

4. Section 118(f)(1)(D)

This subsection asks for information on the methods and options available for reducing children's exposure to environmental sources of lead. Chapter IX ("Methods and Alternatives for Reducing Environmental Lead Exposure for Young Children and Related Risk Groups") addresses this issue. Abatement methods include primary as well as secondary measures. Primary abatement refers to reducing or eliminating lead's entrance into pathways by which people are

36

exposed; secondary abatement refers to ways of dealing with lead after it has already entered the environment or humans. Biological approaches such as improved nutrition may fall into either of these two categories, depending on whether they are intended primarily as prophylactic or treatment measures. Extra-environmental approaches to prevention (e.g., legal actions and strictures) are also discussed.

Here are some key points on the abatement of childhood lead exposure and poisoning :

- Efforts in the United States to remove or reduce human lead exposure have produced notable successes as well as notable failures.
- Effective primary lead abatement measures have included EPA's phase-down regulations for gasoline lead, EPA's national ambient air quality standard for lead, and cooperative actions between the Food and Drug Administration and the food industry to reduce lead in food.
- A number of new initiatives are being implemented by EPA to reduce lead in the drinking water of children and other population segments. Of particular interest is water as it comes from the tap not only in homes but in public facilities such as kindergartens and elementary schools. The schools, in particular, present special exposure characteristics that have not yet been adequately assessed.
- Existing leaded paint in U.S. housing and public buildings remains an untouched and enormously serious problem despite some regulatory action in the 1970s to limit further input of new leaded paint to the environment. For this source, corrective actions have been a clear failure.
- Lead in dust and soil also remains a potentially serious exposure source, and remediation attempts have been unsuccessful.
- Secondary prevention measures in the form of U.S. lead screening programs for children at high risk still appear to require improved standardization of screening methodology (criteria for populations, measurement techniques, data collection, data reporting and statistical analysis) and central coordination.
- The effectiveness of screening children for lead poisoning is well demonstrated in terms of deferred or averted medical interventions, and in most settings is quite cost-effective.
- Extra-environmental measures, such as comprehensive good nutrition programs, have a role in mitigation of lead toxicity, but they cannot be used as substitutes for initiatives to reduce lead in the environment.

- At present, legal sanctions do not appear to be very effective; to be effective, sanctions have to be both meaningful and rigidly enforced. So long as it is cheaper to pay a fine than to remove lead from the child's environment, little progress is likely to be made on this front.
- The "easiest" steps to lead abatement have already been taken or are being taken. These steps, not surprisingly, have involved reducing lead in large-scale sources, such as gasoline and food, with more-or-less centralized distribution mechanisms.
- Enormous masses of lead remain in housing and public buildings, along with large amounts of lead in dust and soil. If these highly dispersed sources are to be abated, huge efforts will be required.

5. Section 118(f)(2)

Chapter X ("A Review of Environmental Releases of Lead as Evaluated under Superfund") was prepared by the U.S. Environmental Protection Agency (EPA) in response to Section 118(f)(2). The National Priorities List (NPL) of September 30, 1987, was reviewed to identify those sites containing lead. Of the 457 sites, 307 have lead as an identified contaminant and 174 have an observed release of lead to air, to surface water, or to ground water. In addition to describing facilities and lead releases for which remedial action was designed under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), EPA has also gathered data for an urban, non-CERCLA site in Boston where children are exposed to soil contaminated by lead-based paint. This site scored 3.56 under the current Hazard Ranking System (HRS). (The minimum HRS score needed for a site's listing on the NPL is 28.5.) Revisions of the HRS by EPA could change the urban site's score, depending on what revisions are made.

B. SUMMARY OF REPORT RECOMMENDATIONS

The report concludes with Chapter XI ("Lead Exposure and Toxicity in Children and Other Related Groups in the United States: Information Gaps, Research Needs, and Report Recommendations"), an overview of information gaps, research needs, and recommendations. Of key importance are the various general and specific recommendations of the report.

38

In view of the multiple sources of lead exposure, an attack on the problem of childhood lead poisoning in the United States must be integrated and coordinated, if it is to be effective. In addition, such an attack must incorporate well-defined goals so that its progress can be measured. For example, the lead exposure of children and fetuses must be monitored and assessed in a systematic manner if efforts to reduce their exposure are to succeed. A comprehensive attack on the lead problem in the United States should not preclude focused efforts by Federal, State, or local agencies with existing statutory authorities to deal with different facets of the same problem. Indeed, it is important that all relevant agencies continue to respond to this important public health problem, but they should do so with an awareness of how their separate actions relate to the goals of a comprehensive attack.

Specific recommendations, by category, are summarized below:

- Coordinated efforts to reduce lead levels in sources that remain as major causes of lead toxicity, particularly paint and dust/soil lead, are strongly recommended.

- Scientific assessments of lead levels in these sources, through strengthening of existing programs to monitor environmental levels of lead, should accompany removal/reduction efforts.

- Major improvements in the collection, interpretation, and dissemination of environmental lead data on a national level are needed. In particular, lead screening data should be compiled in a uniform manner on a nationwide basis.

- Precise and sensitive methodologies for environmental monitoring and *in situ* measurement of lead concentrations in various media are required.

- An integrated assessment of *all* exposure sources for children is required, including those that are obvious and others that are not. Attention should be given to the lead exposure of children away from the home: paint lead, dust/soil lead, and lead in drinking water in schools, day-care centers, custodial care institutions, and similar sites. Particular attention should be given to the investigation of lead leaching into the drinking water of children in schools.

- The report strongly recommends that lead abatement initiatives include careful consideration of lead movement to avoid simply shifting the lead problem from one part of the environment to another.

- The report strongly recommends that much more attention be paid to exposure of the fetus with screening of Pb-8 levels in all high-risk pregnant women.

- Key initiatives recommended by the American Academy of Pediatrics (1987) should be adopted. These initiatives include screening of every child in the United States at risk of exposure to lead.

- The report recommends a careful examination of the role of improved nutrition in ameliorating lead toxicity.

- Continuing large-scale assessments of lead burdens in children, including further national surveys and more regionally focused studies are required.

- Continued support should be given to the highly productive prospective epidemiological studies now under way and to the development and refinement of metabolic models that are used to examine the quantitative relationship between source-specific lead exposure levels and the resulting lead levels in blood or other body compartments.

C. ISSUES, DIRECTIONS, AND THE FUTURE OF THE LEAD PROBLEM

1. Issues

A number of key scientific issues concerning lead as a major health problem are of special concern for the establishment of public health policy in the United States. These issues include:

- The Indestructibility of the Problem. As an element, inorganic lead cannot be processed by current technology and destroyed. It will continue to be a potential problem in some form forever.

- The Relative Non-Transferability of the Problem. Lead cannot be easily shifted from a hazardous setting to a nonhazardous setting without some concomitant increased potential risk elsewhere. Once removed from its geologically bound forms by human activities, lead poses a toxic threat for which there are no natural defense mechanisms.

- The Environmental Accumulation Factor. Lead accumulates indefinitely in the environment so long as input continues -- no matter in how small a quantity.

- The Human Body Accumulation Factor. The human body accumulates lead over the individual's active lifetime and does so even with "small" intakes from common sources. For hazards to exist, major exposures at given points in time need not occur.

- The Risk Population Accumulation Factor. Estimates of exposure and toxicity based on data from particular points in time, such as the estimates provided in this report, greatly understate the cumulative risk for a population posed by a uniquely persistent and pervasive pollutant such as lead. This cumulative toll over extended time is of much greater magnitude, and hence concern, than the prevalence or total exposure estimates for any given year.

    (a) An individual fetus is never counted more than once in any survey examining populations. In the absence of effective abatement of lead exposure, the estimate of 400,000 individual fetuses at risk for lead toxicity in a single year becomes 4 million individual fetuses in 10 years, or 20 million in 50 years, of lead exposure.

    (b) Within a given time period, successive sets of preschool children are likely to move into the same housing unit, particularly in the case of deteriorated inner-city tenant housing. Thus, the number of infants and toddlers at risk for the exposure associated with such conditions (especially paint and dust/soil lead) is much greater than the number of deteriorated houses. If one assumes 3 to 5 years as the average period of residency, then perhaps 10 times as many children would be exposed to such conditions over a 30- to 50-year period.

- The Pervasiveness of the Problem. As a pervasive toxicant, lead is shown in this report to affect totals of children that are high in all socioeconomic/demographic strata. The U.S. lead problem is not simply a problem of a generally neglected segment of society. At present, little or no margin of safety exists between existing Pb-B levels in large segments of the U.S. population and those levels associated with toxicity risk.

- Absence of a Truly Optimal Blood Lead Level. As a toxicant serving no known physiological requirement, the presence of lead at any level in the body is less than optimal. Current average Pb-B levels in some U.S. population segments are 15- to 30-fold higher that the theoretical value of 0.5 µg/dl calculated for early, pre-industrial humans.

2. Directions and Future of the Lead Problem

At the same time that progress is being made to reduce some sources of lead toxicity, scientific determinations of what constitute "safe" levels of lead exposure are concurrently declining even further. Thus, increasing percentages of young children and pregnant women fall into the "at-risk" category as permissible exposure limits are revised downward. Accompanying these increases is the growing dilemma of how to deal effectively with such a widespread public health problem. Since hospitalization and medical treatment of individuals with Pb-B levels below approximately 25 µg/dl is neither appropriate nor even feasible, the only available option is to eliminate or reduce the lead in the environment.

In large measure, the more tractable part of the lead abatement effort in the United States is already underway, because the reduction of lead in gasoline, food, and drinking water is amenable to centralized control strategies. Lead in old paint, dust and soil, however, is pervasive and dispersed, and fundamentally different approaches to abatement will be needed. If the Nation is to solve these difficult facets of the lead problem, society must make a strong effort to do so. Without this effort, large numbers of young children in present and future generations will continue to be exposed to persistent and massive sources of lead in their environment.

PART 2

I. REPORT FINDINGS, CONCLUSIONS, AND OVERVIEW

This report responds systematically to the explicit and implicit directives of Section 118(f) of the 1986 Superfund Amendments and Reauthorization Act (SARA). These directives required not only a critical presentation of scientific and public health information on childhood lead exposure and toxicity, but also an examination of these topics in ways new to the scientific and public health communities. To accomplish these objectives, this report has assessed and integrated disparate blocks of information to provide a coherent response.

The lead problem in the United States can be expressed in a simple statement: Lead is potentially toxic wherever it is found, and it is found everywhere. In large measure, this report is an expansion and a quantification of this statement.

This report addresses the adverse biological responses to lead. At times, some have claimed that lead is an essential element, that is, a physiological nutrient. The U.S. Environmental Protection Agency (U.S. EPA, 1986a) examined this issue through a special committee of independent scientific experts, who concluded that the evidence for lead essentially is inconclusive and that there is no evidence that the human body requires lead.

It is not within the scope or purpose of this report to provide an exhaustive review of all available data. For a comprehensive review and assessment of this type, the reader is referred to EPA's four-volume document Air Quality Criteria for Lead (U.S. EPA, 1986a). Original citations and their discussion in this report deal with specific key issues. A number of references in the present report are made to internal documents and unpublished analyses prepared by the Federal agencies that assisted in providing important data.

This chapter is divided into three major sections: (1) a summary of background material from the report (presented in Chapters II-IV of Part 3),

(2) a summary of main findings in the body of the report (contained in Chapters V-XI of Part 3), and (3) conclusions and overview drawn from the report.

A. BACKGROUND INFORMATION TO THE REPORT

The background chapters in Part 3 are themselves summaries of the topics that underlie the preparation of this document. Therefore, this section will address matters of interpretation and overview.

1. Historical Perspectives and Discussion of Important Issues

Chapter II provides both an introduction to lead as a toxicant with a long history of known adverse effects and a discussion of the issues and terms that frame the main body of the report. With respect to the latter, specific attention is given to populations at risk for lead exposure and toxicity, characterization of their lead exposure, the various effects of lead on child health, and the persistence of such effects during the course of development.

Chapter II presents the case that lead is a lingering and pervasive pollutant. Lead adversely affects human populations in many ways and, potentially, in the absence of effective abatement measures, for many years. Once released from natural ores for economic reasons, this element began to accumulate in humans as well as in the environment. Estimates cited in Chapter II indicate that 3-4 million tons of lead as paint, and similar amounts for deposited lead from atmospheric fallout, remain as two examples of persistent pollution (U.S. EPA, 1986a; NRC, 1980). The absence of a biological barrier to lead uptake by the human fetus and the adverse effects induced by such uptake indicate that lead exposure was probably not encountered in early human evolution and that protective mechanisms did not, therefore, develop.

Young children and fetuses are identified as the segments of the U.S. population at most risk for elevated lead poisoning. Pregnant women are identified as a surrogate risk group by virtue of the danger to the fetus. There are other risk populations for lead, of course, including lead workers. However, the groups discussed in this report are in keeping with Section 118(f) of SARA.

These two risk groups, young (especially preschool age) children and the developing fetus in pregnant women, encounter lead from a variety of sources. Six of these sources or source types are of special concern: lead in paint, lead from combusted gasoline, lead from stationary sources of emission, lead in dust/soil, and lead in both drinking water and food. The interactive relationships between human populations and lead sources is complex. It is also the case in the United States that the relative importance of different source categories depends on geographic and demographic/socioeconomic factors. For example, in inner-city areas of older U.S. cities, leaded paint is a major source, as graphically represented in Figure I-1, a map showing the density of pre-1940 housing in the United States. In remote areas of the nation, however, operations such as primary lead smelters may be of most immediate concern.

The relative intensities and time frames for exposures also vary. A small paint chip containing 50% lead as dry film (500,000 parts per million [ppm]), when ingested by a toddler, will most likely produce acute poisoning. However, exposure to lead paint chips is likely to be intermittent. By contrast, chronic exposure of children to levels of lead in dietary media that are far below 500,000 ppm may produce a cluster of chronic adverse effects. In the case of chronic oral intake, the concentration of lead is lower, but the amount of the medium (e.g., food, beverages, tap water) consumed over time is much greater than for a paint chip.

Exposure of risk populations to lead is assessed in several ways. One can measure the level of lead in some exposure medium, such as air or drinking water. This is environmental or external monitoring. Biological or internal monitoring measures the amount of lead actually entering various body tissues or compartments. The most common method of biological monitoring is the measurement of the level of lead in blood (Pb-B). Other measures, such as lead in teeth or lead in bone, are also used to index exposure, but reflect somewhat different toxicokinetic aspects of lead exposure.

2. Lead Metabolism and its Relationship to Exposure, Risk Population Identification and Adverse Health Effects

Chapter III is concerned with lead metabolism in humans. Metabolism, also termed pharmacokinetics, toxicokinetics, or biokinetics, describes the various integrated processes that control the intake, absorption, distribution to

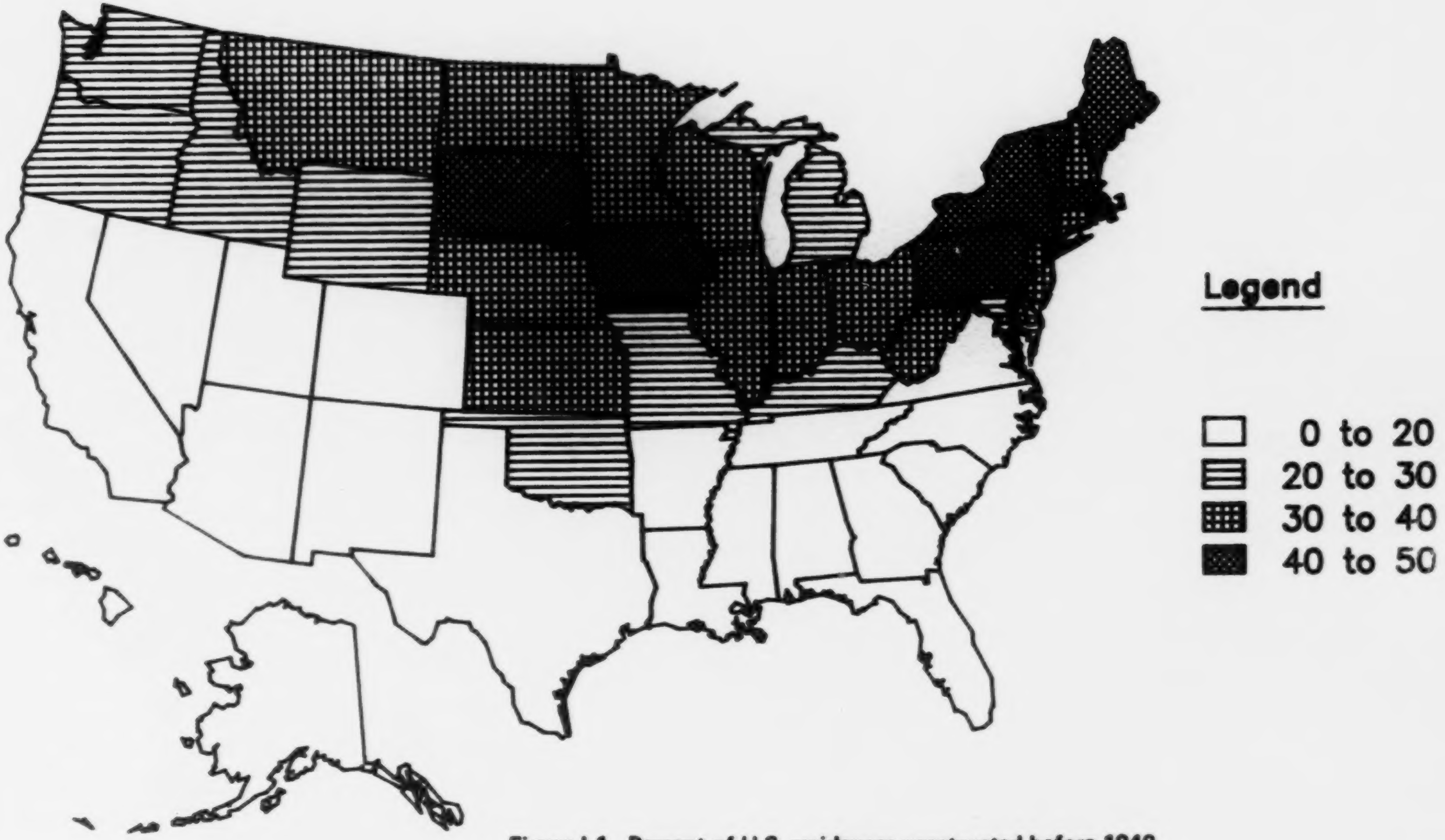

Legend
☐ 0 to 20
▤ 20 to 30
▦ 30 to 40
▩ 40 to 50

Figure I-1. Percent of U.S. residences constructed before 1940.

Source: U.S. Bureau of Census (1986).

tissues, and retention or excretion of lead within the body. Of particular interest are those metabolic aspects that contribute to: (1) the nature of lead exposure and toxicity; (2) identification of those subsets of the U.S. general population at heightened risk for exposure/toxicity; and (3) the metabolic underpinning of the various biological indicators of systemic lead exposure and toxicity.

A general principle of toxicology is that the toxicity of a substance is a function of where the toxic agent goes within the body and how fast it goes there. In addition to following this general relationship, lead has some specific biological properties that merit discussion. In summary:

(1) Lead accumulates internally in humans, and this property poses difficulty for quantifying dose-effect relationships and the temporal pattern of a subject's lead exposure.
(2) The cumulative nature of lead exposure requires that attention be paid to low levels of lead intake/uptake; low levels in media add up to significant exposure over time.
(3) Physiological stress or events such as pregnancy and nursing may mobilize lead from storage sites in the body.
(4) With body accumulation over time, proportionately more of the lead in blood is from internal sources and not only from ongoing intake/uptake.

A number of metabolic criteria serve to define young children and the fetus as subjects at increased risk for lead exposure and toxicity. Young children ingest and absorb a larger amount of lead per unit body measure than do adults. Children also retain a larger fraction of absorbed lead than do adults. Furthermore, children are not as efficient at sequestering circulating lead in their bones, and a higher fraction of total body lead burden is available to exert toxic effects in various target organs. In children, basal metabolism and rates of respiration are higher than in adults, and these act to enhance lead uptake and toxicity risk. Such increased risk occurs at the precise time when children are not only most in need of optimal nutrition, but when nutrient deficiencies are likely to occur. It has been shown in animals and humans, and especially in young children, that nutrient status affects the rate of lead absorption and retention. Deficiencies in iron, calcium, and other elements, commonly encountered in young children, will enhance lead uptake/retention and increase the toxicity risk.

For the fetus, there is no effective metabolic or anatomical barrier to lead uptake. Consequently, there is no protection at crucial time periods when important organ systems such as the central nervous system are forming.

Blood lead is recognized as a relatively short-term measure of lead exposure compared to measures made in, for example, mineralizing or keratinizing tissue. However, serial Pb-B levels are generally correlated over time. Lead levels in accumulating tissues such as bone and teeth better integrate cumulative exposure to lead, but such values are not easily obtained. Thus, Pb-B levels are still considered to be the most useful and practical monitor of exposure, whatever their shortcomings in certain circumstances. Perhaps the best approach to exposure monitoring, in terms of practicality and human toxicological importance, would be the simultaneous monitoring of Pb-B levels and bone lead accumulation by means of special *in vivo* techniques now under development.

3. Adverse Prenatal and Postnatal Effects of Lead in Children: Relationship to Public Health Risk and Their Relative Persistence

Chapter IV provides a concise summary of the health effects of lead, with emphasis on both their significance and their persistence in children. Particular attention is given to those adverse health effects associated with chronic lead exposure at rather low levels.

The various adverse health effects of lead discussed in Chapter IV include *in utero* toxicity as well as postnatal effects in young children. Of particular interest are effects on the central nervous system (CNS), the heme biosynthesis pathway, the vitamin D hormonal system, and various facets of growth and maturation.

Data on chronic injury to the developing human CNS come primarily from early clinical observations, cross-sectional epidemiologic investigations, and more recently, longitudinal or prospective epidemiologic studies. Ongoing prospective studies in various areas of the world are particularly noteworthy because of their methodological strengths. As a group, they provide consistent evidence of developmental impairments at levels of exposure that were generally considered acceptable until recently. Specifically, these studies show significant decrements on the Mental Development Index of the Bayley Scales of Infant Development for as long as two years after birth, thus far. Preliminary data from one study suggest that effects are also evident at five years of age in the form of decreased cognitive abilities associated with blood lead levels three years earlier. In the case of effects related to fetal exposure, blood lead concentrations of 10 to 15 µg/dl, and possibly lower, constitute a level of concern.

When the various cross-sectional studies of young children for neurobehavioral effects of lead are considered together, certain effects such as IQ decrements are clearly seen to be associated with higher levels of lead contact, although some of these studies may have been confounded by factors such as social class. However, recent evidence from well-controlled cross-sectional studies document IQ deficits at Pb-B levels below 25 µg/dl. Other studies also point to reductions in hearing acuity and various neuroelectrophysiological alterations associated with low levels of exposure.

A number of low-level lead effects have been documented in target organs other than the CNS. A concise account of the many ways in which lead can impair the heme biosynthesis pathway and a cascade of heme impairments in various organs and systems is presented in Chapter IV and graphically depicted in Figure IV-2. Also described is the general impairment of calcium pathways through lead-induced disturbance of vitamin D hormone function.

Of particular importance are recent results from prospective studies documenting reductions in gestational age and birth weight associated with fetal exposure levels of <15 µg/dl, levels that are not uncommon in women of child-bearing age in the United States. Other analyses indicate that indices of growth and stature may be reduced in relation to lead exposure in children up to about 8 years of age.

The public health implications of these various findings, particularly longitudinal results showing developmental deficits, are of concern. In the United States, periodic statements of the U.S. Centers for Disease Control (CDC) provide a public health consensus on the level of unacceptable lead exposure risk in children. In its latest statement, CDC (1985) indicated that a Pb-B level of ≥25 µg/dl and an erythrocyte protoporphyrin (EP) value of ≥35 µg/dl provide a practical screening measure of lead toxicity, based on logistical and methodological considerations as well as health concerns known at that time. The World Health Organization (WHO, 1986), in its draft report on air quality guidelines, identified 20 µg/dl as the Pb-B level of concern;

and EPA's Clean Air Scientific Advisory Committee (Lippmann, 1986) has indicated that levels of 10 to 15 µg/dl can be argued as being associated with various adverse health effects.

A further issue specifically identified in Section 118(f) is the persistence of the adverse health effects of lead. Lead-induced injuries to the central nervous system of children are generally considered to be largely irreversible (American Academy of Pediatrics, 1987). Table I-1 identifies neurological and other features of childhood lead toxicity that reflect persisting effects, based on available evidence from the clinical and epidemiological literature.

In the case of injuries to the central nervous system of children, this persistence is seen in continued deficits in neurobehavioral development associated with prenatal and/or early postnatal lead exposure and IQ deficits and altered reaction time in older children who were exposed to lead up to 6 years earlier. Recent preliminary evidence suggests that cognitive deficits at about five years of age are linked to lead exposure at two years of age.

Even when certain impairments are biologically reversible, continuing exposure conditions can result in de facto persisting effects. The best example of this circumstance is the lead-induced derangement of heme biosynthesis, including reduced heme formation. These effects are technically reversible, but many inner-city, poor children cannot escape the exposure causing these effects.

A key question in the persistence of lead toxicity is how long an effect need persist before permanent long-lasting effects on systems or functions occur. These latter effects may now be occurring undetected. Prudence would dictate that the longer an effect persists in the body, the more one should consider the likelihood that it will potentially cause long-lasting consequences.

B. MAIN REPORT: QUANTITATIVE EXAMINATION OF LEAD EXPOSURE AND TOXICITY RISK IN CHILDREN AND PREGNANT WOMEN AND STRATEGIES FOR ABATEMENT

Following a discussion of adverse health effects and their persistence in Chapter IV, detailed responses to other directives of Section 118(f) of SARA are presented in the latter part of the main report, Chapters V-XI. Section 118(f) directed an examination and determination of (1) numbers of children

TABLE I-1. RELATIVE PERSISTENCE OF ADVERSE HEALTH EFFECTS IN INFANTS AND CHILDREN[a]

Adverse Effect	Length of Study Period(s)	Comments
Reduced gestational age and birth weight	Birth onwards, developmental deficits. Pregnant mothers enrolled prior to delivery, offspring followed up to 24 months postnatally, thus far.	Major predictors for persisting, later developmental problems.
Deficits in Bayley Mental Development Index	Up to 24 months thus far.	Early neurobehavioral tests assess functional health of nervous system in infants.
Preliminary indications of deficits in McCarthy Scales performance at 5 years of age	Relationship observed over 3-year interval thus far.	Effect related to postnatal exposure at 2 years of age.
IQ deficits in school-age children and other measures	With higher Pb-B levels, IQ deficits persist. Low levels show several years of persistence w/Pb-dentine; may not be detectable with Pb-B. Reaction time effects appear to persist up to 6 yrs post-exposure, using Pb-tooth as index.	Any persistence in IQ deficits carries risks for other psychosocial effects. Good evidence for persistence of cognitive deficits stems from nonhuman primate data showing related but irreversible impairment of learning acquisition. Ongoing prospective studies will provide key to many questions.
Neurophysiological disturbances	Five years after most Pb-vulnerable period, effects remain on CNS sensory pathways depending on conditioning paradigm employed.	Passive conditioning stimulation approaches show persistence up to 2 years; no persistence at 5 years. Active conditioning task testing not done originally.
EP elevations	Elevations persist with both external exposure and endogenous (bone) lead release.	Cascade of effects from body heme pool disturbances (see Figure IV-2) include neurological development.

[a]Source: U.S. EPA (1986a) and Davis and Svendsgard (1987), with updating.

exposed to lead at unacceptable levels and characterized by geographic area, (2) numbers of children exposed to lead and ranked by source(s) of exposure, and (3) methods and options for how such numbers may be reduced. Section 118(f) also directed an evaluation of the risk to children from lead at Superfund sites using EPA's Hazard Ranking System (HRS), and EPA has prepared a separate response to this portion of the statute (Chapter X). The report ends with a summarizing list of data gaps, research needs, and recommendations in Chapter XI.

1. Ranking of Lead-Exposed Children by Geographic Area

Chapter V presents a detailed discussion of the attempted identification and ranking of lead-exposed children by areas of the United States. As one means of achieving this, the information in Chapter V has been divided into three sections: (1) estimated numbers of lead-exposed children at varying toxicity risks in terms of Pb-B levels and within some level of geographic differentiation; (2) actual counts of lead-exposed children meeting or exceeding current minimal criteria for unacceptable exposure within screening programs in various U.S. states, counties, and cities; and (3) actual 1980 U.S. Bureau of Census counts of children in each of the 318 U.S. Standard Metropolitan Statistical Areas (SMSAs) having a source-specific exposure. This specifically includes young children exposed to high levels of paint lead in housing of various ages.

The first section of Chapter V, children in defined areas, describes estimation efforts that contain two components: (1) 1980 census-based stratification of young children into various socioeconomic/demographic categories and applied to the child population aged 6 months to 5 years in 1984, and (2) projected prevalences of Pb-B levels at certain criterion values to produce estimates of children in each of the given strata whose Pb-B levels will lie above these values. The most recent reference year for which reliable and actual enumerations could be obtained was 1984. For each stratum of children where Pb-B prevalences could be provided, every attempt was made to furnish a corresponding actual count of children by which the prevalence could be multiplied to yield estimated numbers of children actually exposed to lead at the selected level. Prevalences based upon the original data of the National Health and Nutrition Examination Survey (NHANES II), gathered during 1976-1980, were projected to 1984 using a statistical procedure employed by EPA's Office of Policy Analysis with the assistance of staff of the National Center for Health Statistics.

Projected prevalences were obtained as percentages of children in the United States whose Pb-B levels would be expected to lie above selected criteria levels of 15, 20, and 25 µg/dl. These values were selected for reasons given in Chapter IV. The projected prevalences and the numbers of children taken for analysis are those for the fraction of the total U.S. population that lives in Standard Metropolitan Statistical Areas (SMSAs). This fraction represents up to 80% of the U.S. total child population.

Table I-2 presents data from Chapter V showing (1) the total strata-specific numbers of children from the SMSA fraction of the U.S. population and (2) how many of these are estimated to have Pb-B levels above the criterion value of 15 µg/dl, a level identified as being associated with onset of adverse health effects. The table yields a sum indicating that about 2.4 million black and white U.S. children, or 17% of this SMSA-based child population aged 5 years or less, have Pb-B levels above 15 µg/dl.

Table I-3 shows the numbers of these children in U.S. SMSAs with Pb-B levels above 15, 20, and 25 µg/dl. It corresponds with the numerical estimates at 15 µg/dl in Table I-2, but extends to higher Pb-B levels with fewer strata.

Strata in Tables I-2 and I-3 that might be expected to contain large numbers of lead-exposed (e.g., lower income, inner city, blacks) do indeed tend to have the largest numbers at any reference Pb-B level. However, other strata of children are also of considerable concern because, even though prevalences may be lower in certain strata, the base population numbers are much greater. The resulting numbers in the exposure groups, therefore, are also sizable. Since approximately 75 to 80% of all U.S. children live in SMSAs, a conservative estimate of total number of black and white U.S. children with blood lead above 15 µg/dl would bring the national exposure figure close to 3 million children. If all racial categories are considered, then the total for all U.S. children is probably between 3 and 4 million.

A second basis for estimating numbers of lead-exposed children in an area-specific way is to examine data from the various lead exposure and poisoning screening programs. Such programs have operated and continue to operate in a number of states, cities, and other locales.

Screening programs in the United States began in the early 1970s and have undergone a number of administrative and policy-based changes over the years.

TABLE I-2. ESTIMATED NUMBERS OF CHILDREN, 6 MONTHS TO 5 YEARS OLD, WHO ARE PROJECTED TO EXCEED 15 μg/dl Pb-B, BY FAMILY INCOME AND RACE, IN ALL SMSAs, 1984

Family Income/ Race	Base Population	Inside Central City[a] <1 M	Inside Central City[a] ≥1 M	Not Inside Central City[a] <1 M	Not Inside Central City[a] ≥1 M	Small SMSAs[b]	Total
<$6,000							
White	1,039,600	33,600	93,400	27,400	71,100	59,700	285,200
Black	698,300	78,900	234,900	14,800	44,600	56,600	429,800
Total[c]	1,737,900	112,500	328,300	42,200	115,700	116,300	715,000
$6,000-14,999							
White	2,666,300	43,700	113,000	46,700	120,400	97,800	421,600
Black	793,300	57,100	184,900	17,500	49,900	56,900	366,300
Total[c]	3,459,600	100,800	297,900	64,200	170,300	154,700	787,900
≥$15,000							
White	7,643,900	46,000	124,600	61,100	241,200	96,900	569,800
Black	991,800	41,800	151,000	14,400	64,400	36,300	307,900
Total[c]	8,635,700	87,800	275,600	75,500	305,600	133,300	877,700
National Total[c]	13,840,000[d]	301,100	901,800	181,900	591,600	404,200	2,380,600

[a] SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).

[b] SMSAs with less than 1 million population except Nassau-Suffolk, NY which has more than 1 million but no Central City.

[c] Totals by addition, not estimation.

[d] Includes 6,800 children from small SMSAs who could not be stratified by family income.

TABLE I-3. SUMMARIES OF ESTIMATED NUMBERS OF CHILDREN 6 MONTHS TO 5 YEARS OLD IN ALL SMSAs WHO ARE PROJECTED TO EXCEED SELECTED LEVELS OF BLOOD LEAD, BY URBAN STATUS, 1984

Characteristic	Base Population	Blood Lead Level (μg/dl)		
		>15	>20	>25
In SMSAs ≥1,000,000	7,251,000	1,493,400	459,500	128,200
In Central City	2,886,200	901,800	301,700	86,200
Not In Central City	4,364,800	591,600	157,800	42,000
In SMSAs <1,000,000	3,536,400	483,000	142,400	40,300
In Central City	1,504,800	301,100	93,800	27,500
Not In Central City	2,031,600	181,900	48,600	12,800
In Small SMSAs	3,052,600[a]	404,200	113,600	31,200
National Total	13,840,000	2,380,600	715,500	200,700

[a] Total includes 6,800 children who could not be stratified by income and were not included in estimates for three Pb-B levels.

Originally, the U.S. screening effort was administered by the CDC. At the end of Fiscal Year (FY) 1981, more than 60 programs were operating under CDC. After FY 1981, Federal support was folded into block grants to the various states, and administrative control is now in the hands of state health officials. Given the nature of the state-specific block grant mechanisms and how screening efforts would be funded within them, it is difficult to contrast data from the current programs with earlier programs under CDC.

Section B of Chapter V presents a detailed discussion and tabulation of various past and present screening programs, the numbers of children screened, and the numbers of positive toxicity cases detected. In FY 1981, the last year of CDC management, 535,730 children were screened, with a positive toxicity rate of 4.1% or 21,897 children. In FY 1983, reports from the state agencies indicated that 676,571 children were screened, and 9,317, or 1.6%, had elevated lead exposure of ≥30 μg/dl and EP ≥50 μg/dl.

In the most recent collection of screening data, carried out by ATSDR in December 1986, 785,285 children were screened in about 40 programs. Of these, 11,739 children, or 1.5%, had elevated Pb-B levels that met CDC's toxicity classification. This recent survey data base includes positive toxicity cases that range from 0.3% in four programs to 11.0% for the City of St. Louis program, based mainly on the 1985 CDC criteria but also using, for part of the period, the earlier CDC (1978) criteria (see Section B, Chapter V). The five highest prevalences are 11.0% (St. Louis), 9.0% (Augusta and Savannah, GA), 4.9% (Harrisburg, PA), 3.5% (Washington, DC), and 3.5% (Merrimac Valley, MA [program within the Maternal and Child Health project]). The majority of the risk prevalences were below 2%.

Over the years, various characteristics of the lead exposure screening efforts in different communities have been changing: the definitions of toxicity risk, the administrative organization of the programs, and the level of funding of the programs, among other factors, have varied considerably. These changes make it difficult to determine such factors as estimated time trends for more recent years, actual prevalences now being obtained, and what they mean compared with results of prior program screening. Whatever these difficulties, some points are worth noting.

1. Trends in lead toxicity prevalence rates over time, at least in the years of control by CDC and possibly later, suggest a moderate decline in positive toxicity risk numbers from 1973 onwards, even taking into account changes in risk classifications.

2. Recent changes in the CDC toxicity risk classifications appear to be producing an increase in the number of positive toxicity cases, at least in one major city program.

3. In coming years, changes in toxicity prevalences will presumably represent changes in the criteria for toxicity and declines in Pb-B levels associated to some extent with decreases in environmental lead inputs.

4. Compared with prevalence projections based on NHANES II data, screening results provide generally lower prevalences, even though children in these programs are "high risk" subjects. Reasons for the discrepancies include the high false negative rate for subjects who are first screened for EP, limited coverage of screening programs, and use of clinic contacts versus home visits. In the case of false EP negatives, any elevated Pb-B levels in these children are lost to accounting. This difficulty did not occur in the NHANES II survey, where direct Pb-B testing was done.

5. Communities that find the highest prevalences of toxicity cases among their children may not have an exceptional lead exposure situation. It may simply be that communities with a more systematic approach are turning up the highest rates, a possibility that requires centralized screening administration for investigation.

Section C of Chapter V examines the numbers of children living in SMSAs by the age of their housing. Age of housing, particularly for the oldest category, can be taken as a reasonably good indicator of the level of potential exposure to paint lead (as detailed in Chapter VI). Also examined is the effect of family income on the distribution of young children by housing stock of varying age. The detailed SMSA-by-SMSA numbers of children in the oldest housing, i.e., housing with paint of highest lead content, are tabulated in Section C of Chapter V and in Appendices A, B, and C. Rankings of SMSAs by numbers of children in variably aged housing and by family income are given for all 318 SMSAs in Section C and Appendix D.

The number of children living in pre-1950 housing was obtained from the actual 1980 U.S. Census enumeration. Tabulations in Chapter V (and supporting data in Appendices A-C) indicate that young children are least often found in the most recently constructed housing, i.e., 1970-80 units. This is probably because young families are least likely to be able to afford newer housing, particularly new housing inside central cities.

Table I-4 presents an illustrative housing and income profile for stratified groups of children in the SMSA for Cincinnati, Ohio-Kentucky. As shown in the table, children in families with incomes of $15,000 or more very often constitute the majority of those in each of the "age of housing" categories. Poorer families constitute a small enough proportion of the total population that the highest income group usually predominates in the three "age of housing" categories.

It is especially important to keep in mind that leaded paint in old housing remains as an exposure source for successive waves of young children who occupy such housing. In other words, the count of children at a specific point in time, such as the 1980 census, is actually multiplied manyfold over an extended period if the exposure source remains unabated. The cumulative tally over 3 to 5 decades for infant and toddler exposure in a residence with unabated leaded paint will be at least five- or ten-fold greater than the numbers presented in Chapter V and supporting Appendices.

2. Numbers of Lead-Exposed Children by Lead Source

In Chapter VI, the numbers of children exposed to lead are described in terms of different lead source categories. Since exact counts of children

TABLE I-4. CINCINNATI, OHIO-KENTUCKY - SMSA CENSUS COUNT OF CHILDREN OF ALL RACES, 6 MONTHS TO 5 YEARS OLD, BY FAMILY INCOME, URBAN STATUS, AND AGE OF HOUSING, 1980

Strata	Number of Children				Percent			
	Pre-1950	1950-1969	1970-1980	Total	Pre-1950	1950-1969	1970-1980	Total
In Central City								
<$6,000	4,200	4,300	800	9,300	21.8	35.2	38.1	27.7
$6,000-14,999	4,800	3,400	300	8,500	24.9	27.9	14.3	25.3
$15,000 or more	10,300	4,500	1,000	15,800	53.4	36.9	47.6	47.0
Total	19,300	12,200	2,100	33,600	100.0	100.0	100.0	100.0
Not In Central City								
<$6,000	2,200	2,900	1,900	7,000	8.8	8.7	5.7	7.7
$6,000-14,999	8,000	4,900	4,700	17,600	32.0	14.8	14.2	19.3
$15,000 or more	14,800	25,400	26,600	66,800	59.2	76.5	80.1	73.1
Total	25,000	33,200	33,200	91,400	100.0	100.0	100.0	100.0
Total SMSA								
< $6,000	6,400	7,200	2,700	16,300	14.4	15.9	7.6	13.0
$6,000-14,999	12,800	8,300	5,000	26,100	28.9	18.3	14.2	20.9
$15,000 or more	25,100	29,900	27,600	82,600	56.7	65.9	78.2	66.1
Total	44,300	45,400	35,300	125,000	100.0	100.0	100.0	100.0

TABLE I-5. CATEGORIES OF ESTIMATION METHODS FOR CHILDREN EXPOSED TO LEAD BY SOURCE

Source Category	Level of Precision	Method of Exposure Measurement
1. Lead in paint	Potential exposure	Determination of numbers of children in housing with highest likely lead-paint burdens; complements Chapter V data
	Potential exposure with a better indication of actual exposure risk	Number of children estimated to be in lead-paint housing with deterioration: peeling paint, broken plaster, damage
	Likely actual exposure	Use of a specifically determined prevalence for an NHANES II stratum matching such children; other, regional survey data
2. Lead in gasoline	Potential exposure (Pb-B changes) in a subset of U.S. urban child population	Total number of young children in 100 largest cities of the U.S.
	Actual exposures based on leaded gasoline combustion	Logistic regression analysis to estimate numbers of children falling below selected Pb-B criterion values
3. Lead from stationary sources	Potential exposure	Total of young children in communities within certain proximity of lead operations
	Actual exposure	Prevalence of indicated Pb-Bs at or above some criterion level in actual field studies of stationary sources
4. Lead in dusts and soils	Potential exposure	Summing of potential exposure numbers from the above three categories

(continued on following page)

TABLE I-5. (continued)

Source Category	Level of Precision	Method of Exposure Measurement
	Actual exposure	Summing of corresponding actual exposure numbers from first three actual exposure categories, or use of multimedia regression equations (not possible with present data)
5. Lead in drinking water	Potential exposure	Numbers of young children in homes with either old lead plumbing or with lead solder in new home
	Actual exposure that is measurable but not highest toxicity risk	Numbers of young children in homes with lead levels in drinking water above 20 μg/liter
	Actual exposure at or near toxic levels	Number of children estimated from NHANES II prevalences of projected toxic Pb-B levels (see Chapter VI for description)
6. Lead in food	Potential exposure at or near toxic levels	Tally of children within selected age group
	Actual exposure	Fraction of those potentially exposed children whose food lead intake may raise Pb-B high enough to cause concern

exposed to lead on a source-specific basis do not exist, these numbers had to be estimated. Table I-5 contains an outline of the various estimation strategies. Since Section 118(f) did not specifically define the level of source-specific exposure to be considered, methods of varying levels of precision were used to estimate actual exposure risk. Table I-5 sets forth the approaches by the six major categories: lead in paint, lead from gasoline combustion, stationary source lead, lead in dust/soil, and lead in food and in water. In some cases, numbers of children in proximity to a source are given.

In other cases, actual prevalences of Pb-B levels in a source-specific manner can be used.

The level of estimation error for each of the source categories cannot be stated. In some cases actual counts of individuals are used, but in other cases elements of an estimation analysis are combined, with each having variable and relatively undefined precision. Some estimates are judged to be upper bounds, and others lower bounds for actual values.

For purposes of summary and discussion, each source-based estimation analysis is presented separately.

Summary of Source-Specific Exposures and Ranking of Lead-Exposed Children by Source

The various source-specific estimates of numbers of children having different levels of lead exposure are presented below as summary statements:

a. Children under 7 years old who are potentially exposed to paint surfaces containing lead concentrations of 0.7 mg/cm^2 or higher number about 13.6 million, of whom about 5.9 million live in the oldest, highest paint lead residential units. Of these children, about 4.4 million live in U.S. SMSAs. Children in deteriorated, old housing number 1.8 to 2.0 million.

b. Children living in old and deteriorated housing and estimated to have Pb-B levels above certain selected criterion values due mainly to paint lead exposure amount to: 1.2 million (at >15 µg/dl), 0.5 million (at >20 µg/dl), and 0.2 million (at >25 µg/dl).

c. Children potentially exposed to lead from combusted gasoline and residing in the 100 largest cities of the United States are estimated to total about 5.6 million.

d. Children 13 years old and younger who are sufficiently exposed to gasoline lead to show declines in Pb-B levels in response to the phasedown of leaded gasoline amount to: 1.6 million falling below 15 µg/dl and about 0.6 million falling below 20 µg/dl. These estimates are seen to be unrelated to any specific Pb-B levels.

e. For stationary sites, namely primary and secondary smelters, the potential exposure estimate is about 230,000 children. Of this number, up to 13,000 children will have Pb-B levels above approximately 20 µg/dl.

65

f. For lead in dust and soil, it is necessary to use source-specific numbers for the generators of this source, i.e., paint lead in old housing, lead fallout from leaded gasoline combustion, and stationary emissions. With regard to potential exposure of children, these primary source surrogates for dust and/or soil lead amount to a range of 5.9 to 11.7 million children. Actual exposures of children to soil/dust lead sufficient to raise Pb-B levels to the range of toxicity risk cannot be estimated at this time, but the actual numbers may be considerable.

g. Virtually all children are potentially exposed to some level of lead in drinking water.

h. About 3.8 million children are exposed to residential drinking water containing the proposed EPA level of 20 μg/liter or higher.

i. Children under 6 years old and exposed to lead in residential drinking water at levels high enough to result in toxic Pb-B levels number 241,000 (at >15 μg/dl). Broken down by Pb-B levels, they number 230,000 (at 15 to 30 μg/dl), 11,000 (at 30 to 50 μg/dl), and 100 (at >50 μg/dl).

j. Children of school age have potential exposure to lead in drinking water in school buildings. Numbers cannot be estimated at this time.

k. Essentially all U.S. children of age 5 years or younger may have some level of food lead exposure.

l. The number of children having food lead exposure sufficient to elevate Pb-B concentration to a level of some concern amounts to about 1.0 million as an upper bound. This is an overestimate arising from the fact that it is based on food lead data for the 1970s, when levels were higher than for more recent years.

The above source-specific estimates represent a variety of estimation methodologies, data bases of differing age and specificity, and differing levels of estimate error, much of which remain unquantifiable. Consequently, each source should be considered in terms of relative exposure estimates within the source. Once cannot simply add these numbers together to obtain total source-specific exposures.

Given the above qualifications, it is inadvisable to rigidly rank the importance of the described sources purely on a numerical basis. Other points about these source-specific exposures are discussed later in this chapter. Although it is not possible to rigidly rank source-specific exposure, conclusions may be drawn about their relative impact, as set forth in Section C, Conclusions and Overview.

66

3. Number of Women of Childbearing Age and Pregnant Women

Although not specifically required in Section 118(f), this report includes a quantitative assessment of lead exposure of human fetuses via lead exposure of pregnant women in the U.S. population. *In utero* exposure results in postnatal consequences and therefore is considered within the spirit of Section 118(f) and requires inclusion in this report.

In pregnant women, lead readily crosses the placental barrier and does so early in gestation (see Chapter III). *In utero* exposure, therefore, occurs at periods of development when important organs and organ systems can be affected adversely by lead uptake. Such adverse *in utero* effects have been known for many years and still remain as a public health problem in the form of low-level lead effects, as documented in Chapter IV and in U.S. EPA (1986a).

Based on the evidence cited above, *in utero* exposure appears to produce effects of concern at Pb-B levels of 10 to 15 µg/dl and possibly lower. The implication is that every pregnancy potentially represents a fetus at risk if the mother is found to have a blood lead level of about 10 µg/dl or more. Since pregnant women are not a stable population segment, the total population of women of childbearing age must also be considered when trying to assess the size of this part of the public health problem associated with lead exposure.

The methodology to estimate the numbers of lead exposed women is basically the same as that employed for the numbers of young children projected in Chapter V. For reasons indicated earlier for children, primary attention was placed on that segment of the female population living in SMSAs. After deriving the number of women of childbearing age for 1984, as well as the number of pregnancies for that year (live births plus fetal deaths and legal abortions), the estimated numbers of these women with Pb-B concentrations above four reference blood lead levels (>10, >15, >20, and >25 µg/dl) were calculated. Prevalences for the Pb-B criterion levels were determined by application of logistic regression analysis of the NHANES II data for four race/age categories of women and adjusting for the effects of the phasedown of lead in gasoline.

Table I-6 shows the findings for 1984. About 41,300,000 women were of childbearing age; this represents about 45% of the total female populations in all SMSAs. Of these, about 4,460,000 would be expected to have a Pb-B level

TABLE I-6. ESTIMATED NUMBER OF WOMEN OF CHILDBEARING AGE AND ESTIMATED NUMBER OF PREGNANT WOMEN AND PROJECTED NUMBERS ABOVE FOUR SELECTED Pb-B CRITERION VALUES (µg/dl), BY RACE AND AGE, IN ALL SMSAs, 1984

Race/Age (years)		Base Population	Pb-B (µg/dl)			
			>10	>15	>20	>25
Women in SMSAs[a]						
White	15-19	5,478,000	504,000	27,400	5,500	1,600
	20-44	29,740,000	2,884,800	535,300	119,000	29,700
Black	15-19	1,098,000	90,000	14,300	2,200	500
	20-44	4,984,000	981,800	184,400	34,900	10,000
Total[b]		41,300,000	4,460,600	761,400	161,600	41,800
Pregnant Women in SMSAs[a]						
White	15-19	433,000	39,800	2,200	400	100
	20-44	2,380,000	230,900	42,800	9,500	2,400
Black	15-19	187,000	15,300	2,400	400	100
	20-44	595,000	117,200	22,000	4,200	1,200
Total[b]		3,595,000	403,200	69,400	14,500	3,800

[a] Method of calculating explained in text of Chapter VII.

[b] Totals by addition, not estimation.

above 10 µg/dl. About 9% of the women aged 15 to 44 were pregnant in 1984, and projection of the estimated prevalences yielded about 403,000 pregnant women with Pb-B levels above 10 µg/dl, which signifies that their fetuses were at risk for abnormal prenatal as well as postnatal growth and development (see Section A and Chapter IV).

Since the individuals who are pregnant vary from year to year, the population at risk is constantly changing and is not readily identifiable. In other words, it is not a single, fixed group of pregnant women who constitute a one-time exposure risk to their fetuses. Over a 10-year period, for example, in the absence of effective exposure abatement the cumulative number of individual fetuses at risk would be greater than 4 million, even assuming multiple pregnancies in some women in this pool.

Women of childbearing age, 15 to 44 years, have a smaller uptake of airborne lead than children on a body weight basis, and unlike children, they obtain the major portion of the total body burden of lead from food and water rather than from leaded paint chips, dust, and contaminated soil. Abatement limited to external exposure sources of significance for children may not necessarily achieve comparable improvement of lead exposure in women.

4. The Issue of Low-Level Lead Sources and Aggregate Lead Exposure of Children in the United States

Chapter VI of the main report, which deals with single-source exposures, provides estimated numbers of children exposed to lead in some dominant source or cluster of sources, such as dust and soil. This is possible because of the high concentrations of lead in these sources.

It is not possible, however, to assess the impact of low-level lead sources such as food or water without simultaneously considering other lead inputs that may also be moderate in relative impact, because multiple, low-level inputs can accumulate to a potentially significant aggregate exposure. Lead entering the body from a variety of sources presents a unified toxicological threat that is independent of the source of exposure. With multiple, low-level aggregate intake and uptake, it is essential to have ways to examine changes in levels of these sources as they relate to changes in Pb-B levels. This imperative is the rationale behind considering low-level sources, the subject of Chapter VIII.

The cumulative exposure approach also requires us to examine various parameters associated with different body organs and functions (e.g., lungs versus the gastrointestinal tract) in the same population group or among various groups (e.g., children versus adults). Details of the metabolic factors appear in Chapter III.

The issue of aggregate exposure to lead has both scientific and regulatory policy aspects. With respect to policy, these aspects include the degree of remediation and feasible abatement level possible for various lead sources. For the purposes of illustration, consider a childhood Pb-B level of 25 μg/dl as an index of toxicity risk. If Source A contributes an equivalent of 20 μg/dl, or 80% of this burden, and Source B contributes 5 μg/dl or 20%, then one can remove the major source of the lead, 80% and have left 20% or 5 μg/dl. If this major source is not abatable, but abatement of the minor source is possible, then the latter action is still useful. By doing so, one can drop the Pb-B level from 25 to 20 μg/dl, that is, below the given "toxicity" level selected for this illustration.

Several approaches to the problem of the aggregate impact of lead from a variety of low-level sources are examined in Chapter VIII. National or regional surveys of Pb-B levels would, of course, reflect the total, integrated amount of lead absorbed from all sources across national or regional population groups; such surveys could set baselines for observing changes. Methods for tracing the contributions of specific sources to blood lead need to be better developed, and the impact of lead distributions from cumulative sources (e.g., on young children in a given region) needs to be analyzed. Quantitative metabolic models of lead intake, uptake, and systemic distribution that permit the factoring of all inputs to blood lead, even when such inputs vary across pediatric groups, need to be developed or further refined. Further data on the distribution of lead between the fetus and the mother would be of particular value.

Chapter VIII addresses each of these topics in some detail, with particular emphasis on the use of comprehensive metabolic/kinetic models that would be of use to regulators examining multisource lead inputs to human populations. Such models enable one to consider the incremental inputs of individual sources to blood lead as well as the result of removing or reducing a specific exposure source.

5. Lead Exposure Abatement Strategies and Alternatives

In Chapter IX, exposure control approaches are described in general, and lead exposure control approaches in particular. A basic question underlying such past and ongoing exposure abatement efforts is the degree to which reduction should be attempted. For example, is it enough simply to reduce exposure so that Pb-B levels fall below some official index of toxicity, or should some margin of safety be sought? Questions of this sort shape the full scope of abatement efforts.

Lead exposure prevention can be described along various categorical lines, as presented in Table I-7. Prevention efforts can be defined as either primary or secondary, i.e., direct intervention applied globally prior to identification of health risk, or intervention after the fact of identified health risk.

TABLE I-7. CATEGORICAL TABULATION OF THE ELEMENTS OF PRIMARY AND SECONDARY PREVENTION OF LEAD EXPOSURE IN CHILDREN AND RELATED RISK GROUPS OF THE UNITED STATES

Type of Prevention Method	Components of the Measure
I. Primary	
A. Environmental	1. Lead in paint 2. Lead in ambient air (a) Leaded gasoline combustion (b) Point source emissions 3. Lead in dust/soil 4. Lead in drinking water 5. Lead in foods
B. Environmental/Biological	Source controls augmented by passive community nutrition interventions for calcium and iron
II. Secondary	
A. Environmental	1. Case finding 2. Screening programs 3. Environmental follow-up 4. Event-specific exposure abatement
B. Environmental/Biological	Nutritional assessment and follow-up on *ad hoc* identification basis
C. Extra-environmental	Legal actions and strictures

Primary prevention efforts for lead exposure include purely environmental approaches as well as a combination of environmental and biological approaches. A similar duality can also be used in secondary prevention, with the added use of extra-environmental steps, such as legal actions and strictures. With respect to primary prevention, Chapter IX examines regulatory and other measures on a source-by-source basis, since such have been the avenues by which Federal and other governmental actions have been carried out.

Lead in Paint

Paint lead exposure abatement has been attempted through various governmental actions. In 1977, the Consumer Product Safety Commission (CPSC) reduced the legally allowed level of lead in paints to 0.06% net weight. CPSC has no mandate to control leaded paint produced before this date or to address the problem of leaded paint already in housing. Preexisting leaded paint problems are the responsibility of the Department of Housing and Urban Development (HUD). HUD can only regulate leaded paint in public housing or federally assisted dwellings.

Although HUD had some earlier responsibility over the paint lead problem, the bulk of HUD's activity is now in the form of three specific regulations. These three regulations, now in effect, differ in scope, level of control, and likely effectiveness. For public housing, housing inspections and the finding of children with elevated Pb-B levels are trigger mechanisms for leaded paint abatement. Various FHA programs now require paint lead abatement in FHA-assisted sales or purchases. Refinancing of community-based grant programs, such as Urban Development Action Grants (UDAGs), will require evidence of removal of paint lead exposure problems. At this time, it is not clear what the total impact of these recent HUD regulations will be, since limitations are attached to their scope and applicability.

State and municipal actions for the reduction of exposure to paint lead have consisted of statutes with limited application and have received variable enforcement. Among the states, Massachusetts banned lead in any unit in which young children live, but organized opposition from real estate interests and limited funding for enforcement essentially reduced the measure to one of secondary prevention, that is, intervention only after demonstrated instances of lead poisoning have been found. In Chapter IX, Table IX-4, it can be seen that cities in Massachusetts have tens of thousands of pre-1940 units painted with high lead-content paint, but the multi-year abatement counts total only in the hundreds or less.

Certain U.S. cities took actions against leaded paint sale and use well before Federal or state agencies became involved. In 1951, Baltimore, MD prohibited the use of leaded paint for the interiors of dwellings and, in 1958, required warning labels on cans of such paint. In the early 1970s, Philadelphia implemented a primary prevention ordinance against leaded paint, but more recently, prophylactic removal has been discarded in favor of abatement only after demonstrated toxicity in children.

Lead in Ambient Air

EPA has had regulatory authority over the use of lead in gasoline since 1973. The statutory specifics of such authority are embodied in Sections 108, 109, and 211 of the Clean Air Act. These collectively cover lead emissions to ambient air.

Due to regulations concerned with lead and other pollutants, use of leaded gasoline has been declining since the 1970s, as documented in a number of chapters in this report. With recent phasedown action to reduce the lead content of gasoline to 0.1 grams per liquid gallon by January 1, 1988, there is expected to be, and already has been, a significant impact on body lead burdens in the U.S. population. For example, in the section describing source-specific exposures it is projected that the gasoline lead phasedown will lower Pb-B levels to less than 15 µg/dl in millions of children between now and 1992.

Gasoline lead phasedown also affects further input into ecological compartments, although past depositions (fallout) of lead onto soil from the widespread use of leaded gasoline will remain. Reductions in emissions from stationary sources, with measurable benefits to neighboring communities, have been achieved by the EPA-promulgated ambient air lead standard. In 1978, this standard was made more strict at 1.5 µg lead/m^3 of air; currently, the standard is under consideration for possible further reduction.

Lead in Dust and Soil

The primary prevention measures for exposure to lead-contaminated dust and soil are usually directed at the generators of lead for these sources, that is, lead from paint, gasoline combustion, and stationary emitters. Such measures may reduce or eliminate further inputs from these sources but will not influence amounts already present.

Currently, no particular body of regulatory action seems to be directed at controlling lead in dust and soil, although, as discussed in Chapter X, several Superfund sites containing lead in soil are due for cleanup. Reasons for the absence of specific regulatory action include lack of awareness of the problem, the complexity of the problem, and lack of data for inputs from primary contributors on a site-by-site basis. At present, field studies are needed to provide evidence that "macro" rather than "micro" control strategies are effective means of lead abatement in areas larger than a single home or several homes. Mobility of lead in dust and soil prevents simple extrapolation to a neighborhood or even larger area. Field surveys need also to define the relationship between blood lead and primary sources.

The 1986 Superfund Amendments and Reauthorization Act provided for the funding and establishment of a demonstration project as a mechanism to begin to address the problem of area-wide soil (and dust) lead in urban tracts. In response to this, EPA conducted an experts' workshop on the design and scientific operation of soil lead abatement projects in early April 1987 at Research Triangle Park, N.C. Such matters as methods of environmental monitoring, methods of biological monitoring, and statistical design of the population surveys were discussed. A workshop report is currently in preparation.

The Region I office of EPA, in Boston, MA, with the assistance of the Harvard University School of Public Health recently carried out an examination of soil lead removal options. Their conclusions, at least as applied to Boston, indicate that specific soil lead abatement alternatives are a function of the amount of lead present, the disposal methods available, and relative costs. The draft report is presented as Appendix E and is summarized in Chapter IX.

Lead in Drinking Water

EPA is required by the 1974 Safe Drinking Water Act (SDWA) to set drinking water standards, with two levels of protection spelled out in the legislation. Of interest here are the primary standards for drinking water, which define contaminant levels in terms of maximum contaminant level (MCL) or treatment requirements. MCLs are limits enforceable by law and are to be set as close as possible to maximum contaminant level goals (MCLGs), which are levels essentially determined by the relevant toxicologic and biomedical considerations, independent of feasibility. Congress recently ordered EPA to revise the drinking water standards for various substances as necessary, including that for lead. The current MCL for lead is 50 μg Pb per liter of water (μg Pb/liter). It is expected that the revised standard will be somewhat more stringent, possibly 20 μg Pb/liter or even lower. At 20 μg Pb/liter, EPA estimates that about 20% of the tap water levels for homes on public water systems would exceed the proposed standard.

In addition to the pending rule on lead in drinking water per se, the 1986 amendments to the SDWA ban the use of lead solder and other lead-containing material in plumbing connected to public water supplies. States must enforce the ban by 1988 or be subject to a loss of Federal grant funds.

Since EPA is concerned with tap water lead levels, as well as lead burdens in processed water leaving treatment facilities, the agency must specify the "best available technologies" for preventing the entry of lead into drinking water. Two approaches are those of corrosion control, i.e., treating potable water to raise its pH and alkalinity with lime and sodium hydroxide, and the addition of orthophosphate to develop a protective film inside pipes. Furthermore, EPA is considering the removal of lead service connections and gooseneck connectors in its determination of best available technology. The fraction of the U.S. population that has corrosive drinking water is not precisely known, but such water is common to high-density population areas; thus the number of children involved is rather substantial (Chapters VI and IX).

On the basis of studies from both the United States and Scotland, community water treatment as a primary measure to minimize plumbosolvency is known to reduce exposure for children and other groups. In the United States, Boston water authorities began to reduce corrosivity in the 1970s, because of the density of old housing with lead in plumbing. These efforts considerably reduced the amount of lead in tap water (see U.S. EPA, 1986a).

U.S. EPA (1986b) has estimated that the costs of water treatment to reduce corrosivity would be just 25% of the value of health benefits derived from reducing all exposures to lead in drinking water to less than 20 ppb, that is, a benefit-to-cost ratio of 4:1. Therefore, one would expect that extending similar actions under EPA regulations would have a widespread positive impact on children exposed to lead in drinking water.

Lead in Food

Lead from food and beverages is a significant exposure source for a portion of children, considering the distribution of lead intakes within populations of children (see Chapter VI) and the fact that the entire U.S. child population encounters some lead in food. Therefore, primary prevention measures that limit lead exposure from this universal pathway are important. Regulating lead contamination in foods has been the responsibility of the U.S. Food and Drug Administration (FDA) for several decades. This regulatory control dates back to the appearance of lead-containing pesticide residues on sprayed fruits.

FDA actions from the 1970s onward have been aimed at reducing total lead intake or known significant sources of lead inputs into foods. In 1979, FDA made its goal the reduction of the daily total lead intake by children 1 to 5 years old to less than 100 µg/day. This would be the maximum permissible intake, and not a mean intake. Attention was focused on: (1) establishing permissible lead residues in evaporated milk and evaporated skim milk; (2) setting action levels (guidelines for reduction) for lead in canned infant formulas, canned infant fruit and vegetable juices, and glass-packed infant foods; and (3) establishing action levels in other foods. FDA maintains a program to monitor lead levels in the U.S. food supply, but the program is limited and requires expansion. FDA also monitors and enforces controls on such materials as pottery and food utensils with leachable lead; published reports have documented lead exposure from improperly glazed pottery.

The percentage of food cans that are lead-soldered continues to decline. In 1979 the percentage was very high--over 90%; but 1986 figures are expected to be about 20%. FDA has estimated that about 20% of all dietary lead was from canned foods and that about two-thirds of this was from lead soldering.

Recent data provided to FDA by the National Food Processors Association indicate about a 77% reduction in canned food lead in the period 1980-1985. Note that imported canned foods may still come in lead-soldered cans. The number of cans being imported is not known but may be considerable. Recent FDA surveys, from 1982/1983 to 1984/1985, suggest significant declines in daily dietary lead intake across all age groups and for males and females. On average, the decrements in dietary lead intake amounted to approximately 40%. These surveys involved relatively small samples; they need to be expanded considerably in their coverage.

Nutritional Measures in Primary Prevention of Lead Exposure

Several biological factors can suppress lead uptake by the body or enhance its excretion, particularly nutrients that have well-established interactive relationships with lead uptake and toxicity. Supplemental dietary micro- and macro-nutrients may therefore be used to reduce internal exposure. When used on a prophylactic, community-wide basis, nutritional measures constitute one form of primary prevention. When these factors are exploited on an *ad hoc* basis in children or families where lead poisoning has occurred, their use becomes a secondary prevention measure.

Only a few nutrients can realistically be viewed as having a role in preventive community medicine in high-risk populations. Of particular interest are iron and calcium as nutritional supplements. Numerous studies have shown that calcium status and iron status in young children are both inversely related to the level of lead absorption, that is, as either calcium or iron levels go down, lead levels tend to go up.

The reestablishment of optimal nutrition in high-risk children exposed to any level of lead is not only necessary but particularly effective. However, use of optimal nutrition alone, without environmental abatement measures, is not likely to reduce Pb-B levels sufficiently. For the purposes of reducing lead uptake, nutrition monitoring and maintenance are probably best done in a program of nutritional care, such as the Women, Infants and Children (WIC) nutrition program. The level of funding and other support for such programs obviously determines their potential for reducing net lead exposure. Conversely, poor nutrition in those at high risk will enhance toxicity in that population.

Secondary Exposure Prevention Measures

This topic comprises (1) environmental measures, (2) combined environmental and biological measures (nutrition), and (3) extra-environmental measures. The most representative purely environmental measures are community screening programs and associated efforts to identify and abate specific hazards. Features of the various screening programs and the data derived from them are summarized above and discussed in Chapter V. Here, the focus is on their relative roles as secondary prevention instruments.

The lead screening programs administered by the U.S. Centers for Disease Control resulted in about 4 million children being tested nationwide and about 250,000 children being diagnosed as lead poisoned by various criteria. On the average, the screening programs surveyed only about 30% of the high-risk children. Furthermore, the detection rates for positive toxicity were considerably below those of NHANES II. Case finding and cluster testing, followed by targeted screening, also produce much higher positive response rates.

Early screening and detection of lead exposure and toxicity have no doubt reduced the rates of severe poisoning. For a number of reasons, however, chronic exposure and lower grade toxicity appear to be more persistent. Persistence of these problems is predictable, given the levels and types of lead exposure remaining in the United States.

In 1981, Federal resources for screening were put under the program of the Maternal and Child Health Block Grants to States. Although the States' use of Federal funds for lead screening programs was estimated by one source to have been reduced initially by 25% (Farfel, 1985), a precise figure cannot be readily given since allocations of the block grant funds for particular projects are determined by the States according to their priorities, and data are not systematically collected on these State funding allocation decisions.

The evidence of the national impact of this initial reduction in Federal resources appears to be mixed. While it appears that the total number of screening program units in the nation has decreased from 60 to between 40 and 45 (Chapter V), there is also evidence in some States and localities that the number of children currently being screened has increased since 1981 (CDC, 1982; Public Health Foundation, 1986). However, based on a study using data from the period prior to implementation of the block grants (Schneider and Lavenhar, 1986), it is likely that those areas that choose to decrease the efficiency of their lead screening services can expect to experience increases in the number of children with lead poisoning.

Screening programs, especially those supported at levels that allow blanket screening, are particularly cost-effective. To demonstrate this point, the costs of treating lead-poisoned children who were not detected in earlier screening were compared with the costs of community screening programs. In one report, the cost of repeat admissions to Baltimore hospitals for 19 lead-poisoned children was $141,750, or at least $300,000 in 1986 dollars.

For the 1985-1986 program year, the city of St. Louis listed budgetary support of $303,453 from the city and $100,000 from the State of Missouri for lead screening. Concurrently, agencies in the St. Louis program tested 12,308 children, of whom 1,356 or 11.02% were positive for lead exposure as indexed by blood lead levels. These figures are a low boundary for the number of positive cases, because newer, lower guidelines of CDC were only implemented in mid-1985. For the screening period in which its various components detected 1,356 positives, the St. Louis program cost $403,453 or less than $300 per affected child. In contrast, the estimated average cost, in 1986 dollars, for the Baltimore children requiring multiple hospital admissions, was $16,000 per child. Over an extended period, additional costs in the development and care of these children occur and can be large. The effectiveness of screening children for lead poisoning is well demonstrated in terms of deferred or averted medical interventions, and in most settings is quite cost-effective.

In April 1987, the Committee on Environmental Hazards, American Academy of Pediatrics, issued its "Statement on Childhood Lead Poisoning." It includes this statement:

> "...to achieve early detection of lead poisoning, the Academy recommends that all children in the United States at risk of exposure to lead be screened for lead absorption at approximately 12 months of age.... Furthermore, the Academy recommends follow-up...testing of children judged to be at high risk of lead absorption."

These guidelines from America's pediatric medicine community probably cannot be effectively implemented or coordinated with the current levels or existing type of program support at local, State, and Federal levels.

When cases of toxicity were found in the course of mass screenings for lead poisoning, efforts were routinely made to find the causes. A careful examination of the information on reducing lead exposure by completely or partially removing lead paint clearly shows that, at best, the benefit is debatable. At worst, the problem may be exacerbated. One longitudinal study showed that when children return to "lead abated" structures after hospitalization for treatment, their Pb-B levels invariably returned to unacceptable levels. This is not a case of endogenous reexposure from the release of bone lead, because children with equally high Pb-B levels before such treatment retained lower blood levels when returned to housing already free of leaded paint.

Information has accumulated to show that removal of leaded paint is hazardous to the workers doing the removal and that lead from the paint continues to be hazardous to the occupants because residual material has been moved to other areas that children contact. Several recent studies have shown that lead-poisoned children's exposure was exacerbated in various ways when leaded paint removal was being done or had been done in their homes. One difficulty is the relative mobility of powdery leaded paint, which enters cracks and crevasses, settles on contact surfaces, and readily sticks to children's hands.

Therefore, an effective response to the dust aspect of the problem could well be as important as removing paint film.

Secondary Nutritional Measures

As a secondary prevention method, the approach of combining environmental and biological (metabolic) measures through improved nutrition overlaps that described for primary prevention strategies. In this case, however, nutritional optimization to reduce overall toxicity risk is mainly directed to high-risk segments of the population or to children beginning to show elevations in their Pb-B levels. In addition, nutritional approaches used in this way would probably also require the affected family to take a more active role in monitoring the child's nutrition.

Extra-Environmental Prevention Measures

This report considers legal sanctions as a means of forcing the removal of lead from designated sites where there is documented evidence of lead poisoning. It is not easy to draw conclusions from the available information, but it may be useful to examine the experience of a screening program with a legal component. In its summary of screening activities submitted to ATSDR, the City of St. Louis described its dealings with landlords and others who own housing or public facilities where lead poisoning had been found. The main legal device at the City's disposal for forcing lead removal of leaded paint appears to be the imposition of minor fines. It is not clear that minor fines as legal sanctions have influenced the city's lead screening toxicity rate, which in the most recent survey was 11%, a rate that has remained about the same since 1978. This case does suggest, however, that the continuing high rate of lead toxicity has not resulted in more effective legal measures.

6. A Review of Environmental Releases of Lead Under Superfund

Section 118(f)(2) requires the scoring and evaluation of sites at which children are known to be exposed to environmental sources of lead, using the Hazard Ranking System of the National Priorities List. EPA has both listed and discussed facilities with releases of lead that have already been scored using the Hazard Ranking System; it has also collected data on an area in Boston where children have been exposed to soil contaminated by lead-based paint and perhaps lead from past automotive emissions from combustion of leaded gasoline. It was necessary to obtain these data because no such site had ever been submitted for ranking under the Hazard Ranking System.

The facilities that have been listed or proposed for listing on the National Priorities List have all scored above 28.5, the cutoff point under the Hazard Ranking System for listing on the National Priorities List. All were facilities where lead has been or is being smelted or otherwise processed, and all emit or have emitted lead from such processing. All have significant amounts of lead in the soil from such emissions. EPA has examined records to determine whether there is any evidence of children having been exposed to lead from these sources, since children are not an identified population under the Hazard Ranking System. In some cases, especially around primary smelters, earlier studies had shown that children were indeed exposed to the air emissions from the facility and to the contaminated soil, due mostly to the fallout from air emissions from the facility. Those cases are discussed in Chapter X.

The site in Boston for which EPA has gathered data specifically for scoring under the Hazard Ranking System consists of several residences. These residences are located in areas identified by the city as Emergency Lead Poisoning Areas, where a large number of children have been found to have elevated blood lead levels. Soil around these residences contains high levels of lead, much of it apparently from lead-based paint that has weathered and flaked from the houses into the surrounding soil. Some of the lead has most likely come from past automotive emissions resulting from the burning of leaded gasoline.

Only one residence was scored under the Hazard Ranking System for this report, since the data collected at several houses were similar and would result in approximately the same score. The Hazard Ranking System score for the house selected is 3.56 out of a possible 100. This score is the highest possible score for any of the houses, because of its proximity to a surface water source, and because it is within the range of industrial wells. The direct contact score is not used as part of a site's score for purposes of placing a site on the National Priority List.

The Hazard Ranking System is not a risk assessment of the hazards to be found at a facility. Such an assessment occurs later in the process, after additional data have been gathered, and is used to help determine what sort of risk management action must be taken to reduce the risk from the facility to a reasonable level. The Hazard Ranking System was developed to prioritize actual and potential hazards to public health and the environment from hazardous waste disposal sites. It is used by EPA as a screening and prioritizing tool to determine which sites will be candidates for Superfund financial remedial response. Of necessity, it is applied early in the site evaluation process, before many actual data have been collected, thereby requiring that assumptions be made about the site based on available data and on what is known about similar sites with similar releases.

The criteria for setting priorities are based on relative risk or danger, taking into account the size of the population at risk, the hazard potential of the chemicals or substances found or known to be at the facility, and the potential for contamination of drinking water supplies, all of which are factors present at most hazardous waste disposal sites, but not necessarily present in urban residential settings.

The Superfund Amendments and Reauthorization Act of 1986 directed EPA to modify the Hazard Ranking System so that "to the maximum degree feasible, it accurately assesses the relative degree of risk to human health and environment" posed by sites. EPA was specifically directed to assess human health risks associated with actual or potential surface water contamination, damages from an actual or a threatened release to natural resources that might affect the human food chain, actual or potential ambient air contamination, and those wastes described in Section 3001 of the Resource Conservation and Recovery Act (e.g., fly ash, bottom ash, slag waste, and flue gas emission control waste). EPA was also directed to give high priority to facilities where a release has resulted in closure of drinking water wells or has contaminated a principal drinking water supply.

EPA is in the process of addressing these concerns in its update of the Hazard Ranking System. It expects to propose for comment a new Hazard Ranking System in the summer of 1988.

7. Information Gaps, Research Needs, and Recommendations

In preparing this report, it was apparent that adequate information did not exist to fully answer many questions about childhood lead poisoning in the United States. These gaps in information and the research needed to fill them are summarized below. In addition, recommendations for further action are presented.

Information Gaps

(a) Comprehensive, current, and accurate data on the numbers of children and other risk groups having lead exposure by specific areas of residence do not exist as such. Therefore, this report was confined to using, as best as possible, available data.

(b) There is surprisingly little information on the actual numbers of young children exposed to lead in source-specific ways. Data that do exist are highly variable in their quality and level of detail.

(c) Additional data are needed on the relationships between dust/soil lead and both paint and air lead, especially when air levels are changing. In this report, we could not separate these media-specific exposures in order to derive estimates for separate totals of exposed children.

(d) More specific information is required on the actual distribution of lead concentrations in the tap water of households containing young children as well as lead in water of schools, day-care centers, etc.

(e) Information on dietary lead intakes by infants and toddlers needs to be updated. Information is also needed on the distribution of intakes in the U.S. child population.

(f) Much remains to be learned about the adverse effects of lead in young children and other risk groups.

(g) The area of lead exposure abatement approaches and strategies is plagued by a number of unknowns with respect to both their qualitative and quantitative aspects.

(h) Information is needed on the full range of options, in terms of their technology and costs, for removing paint, dust, and soil lead from the environment.

(i) A lack of knowledge exists about support approaches for any assault on lead exposure. These approaches include maintaining optimal nutrition in risk populations and effective legal measures to enforce compliance with abatement programs.

(j) There is no good data base to assess how changes in the organization of screening programs have affected the scope and effectiveness of these programs in high-risk areas. It is particularly desirable to know the level of undetected toxicity among target populations for screening.

Research Needs

A minimum inventory of research needs in the areas covered by this report includes the following:

(1) Comprehensive studies of the relationship between exposed populations and various sources of lead, and the geographic distribution of these risk populations, are needed for high-level sources, as well as those currently viewed as "background" or "low-level." As part of this effort, census-taking instruments should include survey questions related to environmental exposure to toxicants.

(2) Development of quantitative biokinetic/aggregate uptake models for reliably predicting body lead burdens should be continued. Such models could reduce the need for expensive future population surveys.

(3) Both the scope and number of prospective studies of lead exposure and toxicity in U.S. and other populations should be expanded.

(4) Further research on the relative strengths and shortcomings of biological indicators of systemic exposure, particularly *in vivo* measures of lead accumulation in the mineral tissue of young children are needed. A related need is more research on the relationship between fetal lead exposure and the body burden of lead in pregnant women.

(5) In the difficult area of lead exposure prevention by primary and secondary means, research is required on several fronts:

    (a) Further field studies of the efficacy of lead removal from children's environments at a tract or neighborhood level. Efforts would include before-and-after evaluation of Pb-B levels. EPA-sponsored studies are now underway to address these problems systematically via demonstration projects.

    (b) Field studies of the type detailed above that also permit specific assessment of how such procedures are related to primary contributors, such as paint lead versus urban air fallout of lead. Here, also, the EPA demonstration projects will be helpful, if source-dependent studies are made of these variables.

    (c) Assessment of removal technology for site debridement of paint and related sources. Since paint lead alone constitutes an aggregate burden of millions of tons, removal is not an inconsequential problem. Moving the lead may also mean shifting exposure to another population, if adequate precautions are not taken.

(d) Examination of the efficacy of high-risk lead screening programs both for their scope and effectiveness and for the relationship between key factors (e.g., resources made available, screening methodologies, and mechanisms to report data) and their impact on efficiency of identifying children at risk.

(e) Assessment of the relative costs of effective, if expensive, alternatives to the piecemeal abatement, enforcement, and follow-up approaches that appear to constitute present remedial actions. Would it be less expensive, in human and resource terms, to relocate populations as opposed to allowing them to remain in exposure settings?

(f) Research that explores the feasibility of better indicators of lead exposure for screening purposes, particularly at low levels where, for example, erythrocyte protoporphyrin (EP) is less useful. This permits elucidation of those lower effect levels of lead in children not easily identified by EP testing.

RECOMMENDATIONS

In view of the multiple sources of lead exposure, an attack on the problem of childhood lead poisoning in the United States must be integrated and coordinated if it is to be effective. In addition, such an attack must incorporate well-defined goals so that its progress can be measured. For example, the lead exposure of children and fetuses must be monitored and assessed in a systematic manner if efforts to reduce their exposure are to succeed. A comprehensive attack on the lead problem in the United States should not preclude focused efforts by Federal, state, or local agencies with existing statutory authorities to deal with different facets of the same problem. Indeed, it is important that all relevant agencies continue to respond to this important public health problem, but to do so with an awareness of how their separate actions relate to the goals of a comprehensive attack.

Specific recommendations to support the general objective of eliminating childhood lead poisoning are presented below:

1. Lead in the Environment of Children

(a) Efforts should be implemented to reduce lead levels in sources that remain as major causes of childhood lead toxicity.

(1) Leaded paint continues to cause most of the severe lead poisoning in children in the United States. It has the highest concentration of lead per unit of weight and is the most widespread of the various sources, being found in approximately 21 million pre-1940 homes.

(2) Dust and soil lead, derived from flaking, weathering and chalking paint plus air lead fallout over the years, is a second major source of potential childhood lead exposure.

(3) Drinking water lead is intermediate but highly significant as an exposure source for both children and the fetuses of pregnant women. Food lead also contributes to exposure of children and fetuses.

(4) Lead in drinking water is an example of a controllable exposure source for which state and local agencies should be encouraged to enforce strictly the Federal ban on the use of leaded solder and plumbing materials. A second initiative is the examination of the full scope of lead exposure risk from lead-leaching plumbing and fountains/coolers in schools. Strong efforts should also be made to reduce exposure to lead-based paint and dust/soil lead around homes, schools, and play areas.

(b) Efforts at reducing lead in the environment should be accompanied by scientific assessments of the amounts of lead in each of these sources through strengthening of existing programs that currently attempt such assessment. The largest information gap exists in determining which housing, including public housing, contains leaded paint at hazardous levels. A similar gap exists for information in distributions of soil/dust lead on a regional or smaller area basis. Systematic monitoring of lead exposures from food and water is urgently needed. The Food and Drug Administration should seriously consider increasing support for its Total Diet Study to broaden population coverage. The data currently collected do not yield sufficient information on the high risk strata of the population to support intervention measures.

(c) Use of precise and sensitive methodologies is essential for environmental monitoring of source-specific lead. More sensitive and precise techniques are required for in situ field testing of lead in painted surfaces.

(d) Major improvements in the collection, interpretation, and dissemination of environmental lead data on a national basis are required and recommended to assess the extent of remaining lead contamination and to identify trends. Data from screening programs should be compiled nationally and made uniform so that geographic differences in lead toxicity rates can be determined.

(e) The need to examine fully the extent of lead contamination in *all parts* of the child's environment remains. Emphasis should be placed on examining the presence of lead in elementary schools, day care centers, nurseries, kindergartens, and similar facilities, with particular focus on the lead in drinking water, paint, dust, and soil.

(1) All attempts at source-specific lead reductions in children's environments should be accompanied by an assessment of the long-term effectiveness and efficiency of such actions (see Section 2 of Recommendations also).

(2) Lead is ubiquitous and persistent. Planned reductions of lead in one environmental compartment must be evaluated in terms of impacts on other compartments so that fruitless "shifting" of the problem from one source or medium to another is avoided. For example, when leaded paint or soil is removed from a child's environment, consideration must be given to ultimate safe disposal.

(3) The evidence is strong that *in utero* exposure of the developing fetus occurs at potentially toxic levels in some proportion of the pregnant women of the United States. This at-risk population must be given close attention in terms of assessing and reducing their most significant sources of lead exposure. This should especially include consideration of occupational exposure sources.

(4) Lead pollution is a health issue that involves almost all segments of U.S. society. Extra-environmental or legal measures should be explored to reduce lead levels in the environment by both public and private sectors.

2. Lead in the Bodies of Children

(a) Children are being exposed to and poisoned by lead while environmental lead reduction is underway. There is an urgent need for screening programs at levels sufficient to make a real and measurable impact in the prevention of childhood lead poisoning.

(b) There is a need to maintain screening programs extant in some states that currently identify children at risk from lead exposure at or above 25 μg/dl. There is also a need to develop screening tests that will identify children below 25 μg/dl, since current erythrocyte protoporphyrin (EP) tests, used as an initial screening measure, cannot accurately identify children with blood lead levels below 25 μg/dl.

(c) The 1987 statement of the American Academy of Pediatrics calling for lead screening of all high risk children should be implemented.

(d) *In vivo* cumulative lead screening methods should be used as soon as they become feasible. A quick, accurate, noninvasive screening test would result in greater acceptance by parents, resulting in many more children screened.

(e) Screening should be extended to all high-risk pregnant women, with particular emphasis on urban teenaged pregnant women, and prenatal medical care providers should be involved in this effort.

(f) The prophylactic role of nutrition in the amelioration of systemic lead toxicity should be determined.

(g) Further use should be made of already developed metabolic models and research should be conducted to refine them, so that their ability to predict contributions to the total body burden can be utilized for varying environmental source contributions of known lead levels.

(h) It is recommended that long-term prospective studies of lead's effects on child growth and development be supported through appropriate mechanisms, beginning with the relationship of maternal lead burden to *in utero* toxicity and including children with neurological disabilities and genetic disorders, such as sickle cell anemia.

(i) Nationwide assessments of lead toxicity status in U.S. children on a continuing basis are recommended. Efforts such as the planned NHANES III survey should be supported to maximize the data collected on lead exposure levels. Support should be provided for geographically more focused surveys as well, e.g., at the level of Metropolitan Statistical Areas (MSAs).

C. CONCLUSIONS AND OVERVIEW

1. Lead as a Public Health Issue

Lead poisoning has been identified by the American Academy of Pediatrics as one of the most important toxicological hazards facing young children in the United States. In one form or another, lead poisoning has had this status for many decades.

While severe acute and chronic lead poisoning of children was the typical form of the disease at one time, this hazard is now viewed in terms of a cluster of low-level effects collectively known as the "silent epidemic." It must be remembered, however, that unacceptable numbers of severely poisoned children continue to be identified and treated in clinics and hospitals of the nation's urban areas.

The persistence of lead poisoning in U.S. children belies the basic fact that lead intoxication is a fully preventable disease. The level at which actual prevention is attempted, however, has been defined by the level of public attention and the degree of societal commitment at any given time.

With downward revisions in the definition of unacceptable lead toxicity risk in U.S. children, options for prevention of unacceptable lead exposure likewise have changed. Severe lead poisoning of past years, the only form of the disease then considered important, was often considered as manageable by secondary prevention methods, i.e., by identifying the lead-poisoned child and then providing hospitalization and treatment.

The present concern is with low but still important levels of lead exposures associated with subtle health effects occurring at Pb-B levels of 10 to 25 µg/dl. Hospitalization for treatment by chelation therapy, which is acceptable for children with higher Pb-B levels, is not without risk and potentially undesirable sequelae. It is neither appropriate nor feasible for the treatment of the huge number of children at lower Pb-B levels. Therefore, since no acceptable or feasible medical treatment exists at this time, the only option remaining is the removal of lead from the environment of children. Equally valid concerns can be expressed for options in avoiding fetal exposure in pregnant women at levels above 10 µg/dl.

As a public health issue, lead poisoning involves (a) a persistent, ubiquitous toxicant whose environmental distribution produces (b) unacceptably large numbers of children with unacceptably high body lead burdens that require (c) environmental lead removal or reduction as the only exposure abatement option.

2. Lead in the Environment of Children

It is now recognized that potentially hazardous levels of lead can be found in many of the environmental media or pathways that serve as exposure routes for young children. Lead at potentially significant levels may be found in old paint, dust, soil, drinking water, food, and ambient air. Sites of such exposure of children include the interiors of homes, outside play areas, and kindergarten, day-care, and elementary school facilities. A number of such sources can produce simultaneous exposures, raising the question of how one identifies and then ranks the contributions of lead from each source. By and large, control measures for lead in various media have been historically allocated to various public agencies, directed and guided by media-specific legislation.

Section 118(f) implies in its language that one can readily identify a sole or major source of lead exposure for individual children and that these exposure numbers can then be summed and the total estimates ranked by source. In actuality, this report shows that (a) too many large gaps in data exist to quantify precisely source-specific lead exposure of young children; (b) sizable region-based differences in source-specific exposure would probably be diluted or lost in any national rankings; (c) sources with the highest concentrations of lead may not always have the greatest impact; (d) simultaneous exposures to a number of sources occur; (e) relative contributions of a number of lead sources are changing with time; and (f) it is not possible to rigidly rank exposure by source of lead.

Concern for the health of the public and the need for prudent initiatives must cause Congress and society-at-large to ask what conclusions can be drawn from the data presented in this report. Although some of the estimates presented here have limitations and uncertainties attached to them, they still provide a number of key conclusions about source-specific lead exposure of U.S. children:

(a) Lead in paint and lead in dust/soil have been and will remain as major exposure sources for U.S. children.

(b) Leaded paint remains as a major source because of the huge amount of lead in this form--about 5 million tons or more--and the total dispersal of the source among 21 million individual residential units and large numbers of old public buildings. This is the most important source in terms of reservoirs of lead and ubiquity of exposure. Post 1940 housing can also contain high levels of lead in paint.

(c) Dust and soil remain as major sources of lead exposure for children because of the rather high levels of lead in these media, their ubiquitous distribution, and young children's behavior in relation to this source, i.e., mouthing behavior and ingestion of non-food items.

90

(d) Lead in drinking water is currently a potentially significant source of lead exposure, but one that is now being given more regulatory attention. Drinking water in homes, public schools, kindergartens, day-care centers, etc., may have been contaminated by lead in solder, flux, or other components of plumbing systems and devices supplying potable water.

(e) Significant exposure of children can occur from sources away from the home, such as leaded paint, dust/soil lead, and drinking water lead in day care and school facilities.

(f) The phasedown of lead in gasoline has markedly reduced input from this source to human body burdens, which at one time amounted to 40-50% of the burden reflected in Pb-B levels. The accumulated quantities of soil/dust lead from leaded gasoline combustion and atmospheric fallout will only slowly diminish, however.

(g) Food lead has been reduced as a population-wide source of lead exposure in children, especially for infants and toddlers. It is not clear to what extent further reductions will be possible.

In addition to these purely environmental aspects of source-specific lead exposure, this report also draws attention to the fact that leaded paint is the form of childhood exposure that has been associated with the most severe lead poisonings. Other lead sources, e.g., drinking water or food, are judged to provide a continuum of exposure for the populations at risk and to contribute to generally less severe lead poisonings.

The difficulty in judging source-specific contributions to the total body burden of lead and its consequent toxicity poses problems for source-specific regulatory initiatives and for lead abatement strategies. Which source should be first controlled to provide maximum benefit with the minimum expenditure of societal resources? As the report states, rapid reduction of a small fraction of the total body lead burden to below a risk level may be more desirable than removal of a large fraction of body lead that can only be accomplished at a much slower, costlier pace.

3. Lead in the Bodies of Children

Various sources of lead contribute to total body lead burden in a way that is reflected in the most common index of such a burden, the blood lead value. A measurement such as Pb-B level not only integrates multi-source/multi-media exposure but also defines the degree of toxicity risk to be expected when specific Pb-B levels are encountered. This is termed the dose-effect relationship;

as Pb-B level (dose) rises, so do the number of adverse effects and the severity of any given effect.

A crucial point in the understanding of dose-effect relationships for lead is the propensity of this toxicant to accumulate in target tissues of young children and other risk groups. This insidious characteristic complicates interpretation of a Pb-B value, because the latter represents a combination of not only current lead uptake but release of accumulated lead from lead storage areas such as bone. The possibility that accumulated lead may be abruptly mobilized back into the blood stream is especially important for potentially increased exposure of the fetus in pregnancy.

Blood lead levels can be reliably employed to index early effects from low-level exposure to lead, but extreme care must be used for the collection and measurement of blood lead samples at such low levels.

The report concludes that a definition of unacceptable lead toxicity risk continues to undergo downward revision by the scientific and public health communities. Current information indicates that disturbances in various measures of neurobehavioral development, other neurological indices, developmental milestones, and non-neurological systemic functions are seen at child or maternal Pb-B levels in the range of 10 to 25 μg/dl, and possibly even lower than 10 μg/dl. Strong support for these conclusions continues to be provided by well designed and executed longitudinal studies under way in a number of areas of the United States and elsewhere.

These most recently identified effects, while they potentially affect large fractions of the U.S. child population, should not deflect public attention from the fact that unacceptable numbers of children still are treated for severe lead poisoning in the hospitals of the nation's urban areas. These serious injuries involve the central nervous system and the blood-forming system, and can even constitute threats to life itself.

4. The Extent of Lead Poisoning in Children in the United States

This report describes several approaches to Congressionally mandated assessments of the numbers of young U.S. children having Pb-B levels high enough to pose adverse health risks. Results of these assessments provide a number of key conclusions:

(a) Sufficient methodology exists to attempt at least a stratified national assessment of children with unacceptable Pb-B levels. In the main approach used to define lead exposure in terms of a geographic index, a combination of census counts of young children plus vital statistics data were matched up with projected Pb-B prevalences to yield numbers of children in socioeconomic/demographic strata who have Pb-B levels above selected values.

(b) A crucial result from this assessment is that approximately 2.4 million, or 17%, of black and white children who live in SMSAs are estimated to have had Pb-B levels above 15 µg/dl in the reference year of 1984; the criterion level of 15 µg/dl is judged in the report to be associated with health effects in young children. For the entire nation, this estimated number of children would probably lie between 3 and 4 million if all racial and residential categories were included.

(c) The estimates presented here include children from all socioeconomic strata, e.g., urban/suburban children above the poverty level as well as the expected strata of inner-city children from families in poverty.

(d) Prevalences of elevated Pb-B levels are highest for inner-city, underprivileged black children, while rates for other strata of city children, both black and white, are intermediate. Suburban children above the poverty level have the lowest prevalences.

(e) Total numbers of lead-exposed children in nearly all strata are significantly high. The largest base populations have lower prevalences while the smallest population base has the highest prevalences of elevated Pb-B levels. When combined, the results are large numbers in most strata.

(f) The above approach contains uncertainties that may produce both underestimates in the numbers as well as overestimates. Sources of underestimation include restriction of the estimates to only SMSA-based children. Such individuals represent only 75 to 80% of the entire U.S. child population. The projected estimates are restricted to those groups in the original NHANES II survey, black and white children for whom prevalences could be calculated validly. Therefore, the numbers in the report do not include Hispanic and "Other Race" children. The main source of overestimation is the use of a projection model for Pb-B prevalences that did not include downward changes in food lead in the logistic regression analysis. The projections should therefore be considered as "the best estimates consistent with available scientific data."

(g) The report also examines results of past and present lead screening programs in various communities of the United States. For 1985-1986, ATSDR determined through an extensive survey of operating programs that about 1.5% of screened children were at or above CDC action levels for Pb-B and erythrocyte protoporphyrin (EP), using either earlier or more recent CDC classification schemes.

(h) The prevalences of elevated Pb-B levels obtained through screening data and those for various NHANES II strata are seen to be quite different in the report. This is especially so for those NHANES II strata that can only be compared to the high-risk populations examined in screening areas, i.e., inner-city, underprivileged children.

(i) The report concludes that screening versus stratified national sampling results are different for such reasons as (1) major flaws in the methodology of screening programs (resulting in marked underestimates), (2) nonintensive coverage of the target population, and (3) differences in program protocols among different screening programs with respect to which children should be screened.

(j) The distribution of U.S. SMSA children among residences differing in age and therefore in degree of leaded paint exposure was also examined on an SMSA-by-SMSA basis. In all 318 SMSAs, about 4.4 million children live in the oldest housing (pre-1950) and are therefore exposed to the highest levels of lead in paint. The report concludes that the major socioeconomic category of children in the oldest housing, by far, are those above the poverty level. Leaded paint in old housing is not just a problem for poor families in inner-city areas.

(k) Housing and income results complement the NHANES II projections and total exposed population estimates, in that they also show that many strata of children in exposure risk areas are affected, not just inner-city underprivileged groups.

5. The Problem of In Utero Lead Toxicity: The Extent of Fetal Lead Exposure in the United States

In Chapters III and IV, the report concludes that a strong case is to be made for identification of the human fetus as a high-risk population. Fetal exposure (for which the population of pregnant women is the surrogate risk group) leads to a number of potentially adverse effects *in utero* and these may well persist during postnatal development.

Examination of the extent of lead exposure among fetuses in the United States, via pregnant women's exposure, was carried out for 1984 as the reference year. Projection estimates were employed using the methodology already identified for childhood lead exposures. The important conclusions to be drawn from these findings include:

(a) Over 400,000 fetuses in 1984 were exposed to lead at maternal Pb-Bs above 10 µg/dl, a level associated in recent studies with early developmental effects. Furthermore, over 4 million women of childbearing age were so exposed.

(b) The lead toxicity hazard for the U.S. fetal population is multiplied year after year in the absence of effective lead exposure reductions for the pregnant population. A given fetus is not counted more than once in exposure estimates. Thus, more than 4 million individual fetuses will be at risk for toxic effects over a 10-year period in the absence of effective lead removal or reduction.

(c) *In utero* lead exposure in the United States will continue to increase in importance as a public health issue.

6. Removal or Reduction of Lead in the Child's Environment

The report concludes that a strong case can be made for the necessity of both collective and source-specific efforts to systematically reduce or remove lead from the child's environment. At present, there are millions of young children for whom reduction or removal of environmental lead is the only option for lowering high Pb-B levels. There are, likewise, hundreds of thousands of fetuses in any given year and millions of fetuses over extended time periods who require reduction in lead uptake and concomitant reduction in environmental exposure of the female population of child-bearing age in the United States.

A number of important findings in the report are related to environmental lead removal or reduction. Some of the conclusions to be drawn from these findings include:

(a) Certain regulatory and other control actions have been reasonably successful in recent years in reducing source-specific lead exposures. Notably, the phasedown of lead in gasoline is producing demonstrable and significant reductions in population Pb-B levels.

(b) Reductions of lead in food, particularly for diets of infants and toddlers, have occurred and have had important consequences for the "background" levels of body lead burden.

(c) Lead in drinking water is now being attacked on several regulatory fronts by EPA and other relevant agencies. Such actions include bans on the use of lead solder for plumbing, corrosion control measures in public water supplies, and control of lead exposure from lead leaching into water sources in schools, offices, and homes.

(d) Control of lead in ambient air by means of air quality standards for lead will continue to have a positive impact on reducing lead emissions from stationary sources.

(e) Easily-achieved removal or reduction of lead in paint from residential units or public facilities will probably not occur without highly coordinated efforts. HUD has undertaken a number of new actions for detecting and responding to paint problems in Federally assisted housing, but their effectiveness remains to be documented and their scope is greatly limited. As the report notes, millions of tons of paint lead still persist as a highly dispersed and accessible hazard.

(f) Removal of lead from soils and as dusts parallels removal efforts for paint, in terms of complexity. As the report states, millions of tons of lead are now lingering in soil and as dust. Since soil and dust are pathways of exposure, abatement effectiveness in these cases is ultimately governed by abatements in paint lead and air lead fallout.

7. Future Directions of Lead as a Public Health Problem

With significant downward changes in source-specific amounts of lead that have occurred or will occur, some sources will decline in importance. Simultaneously, other sources such as paint and dust/soil lead will continue to receive more attention and more public calls for action. The "easiest" parts of lead exposure control have been implemented or are being set in place, namely the use of centralized control mechanisms, such as the phasedown in the amount of lead in the nation's gasoline supply and the levels of lead in a basically centralized food supply. Corrosion control measures for the public drinking water supply are a similar example, since a large segment of the U.S. population derives drinking water from public systems.

Definitions of unacceptable lead toxicity risk have been undergoing significant downward revisions in recent years, and further changes downward may occur in the future. The result is that any gains in reducing lead exposure are offset by the progressive redefining of how much further lead exposure should be reduced.

The nation will soon have to face fully the abatement of the most refractory and most costly sources of lead exposure and toxicity: paint lead and dust/soil lead. This will require some hard societal choices. Chief among these choices will be whether remaining and problematic sources will be systematically and effectively controlled or whether large numbers of young children and fetuses will bear persistent and unacceptable quantities of lead in their bodies and tissues.

THE NATURE AND EXTENT OF LEAD POISONING IN CHILDREN IN THE UNITED STATES: A REPORT TO CONGRESS

PART 3

(CHAPTERS II-XI)

THE QUANTITATIVE EXAMINATION OF LEAD EXPOSURE AND TOXICITY IN CHILDREN AND RELATED RISK GROUPS IN THE UNITED STATES: CHARACTERIZATION AND METHODS FOR REDUCTION

PART 3

II. INTRODUCTION AND DISCUSSION OF TERMS AND ISSUES

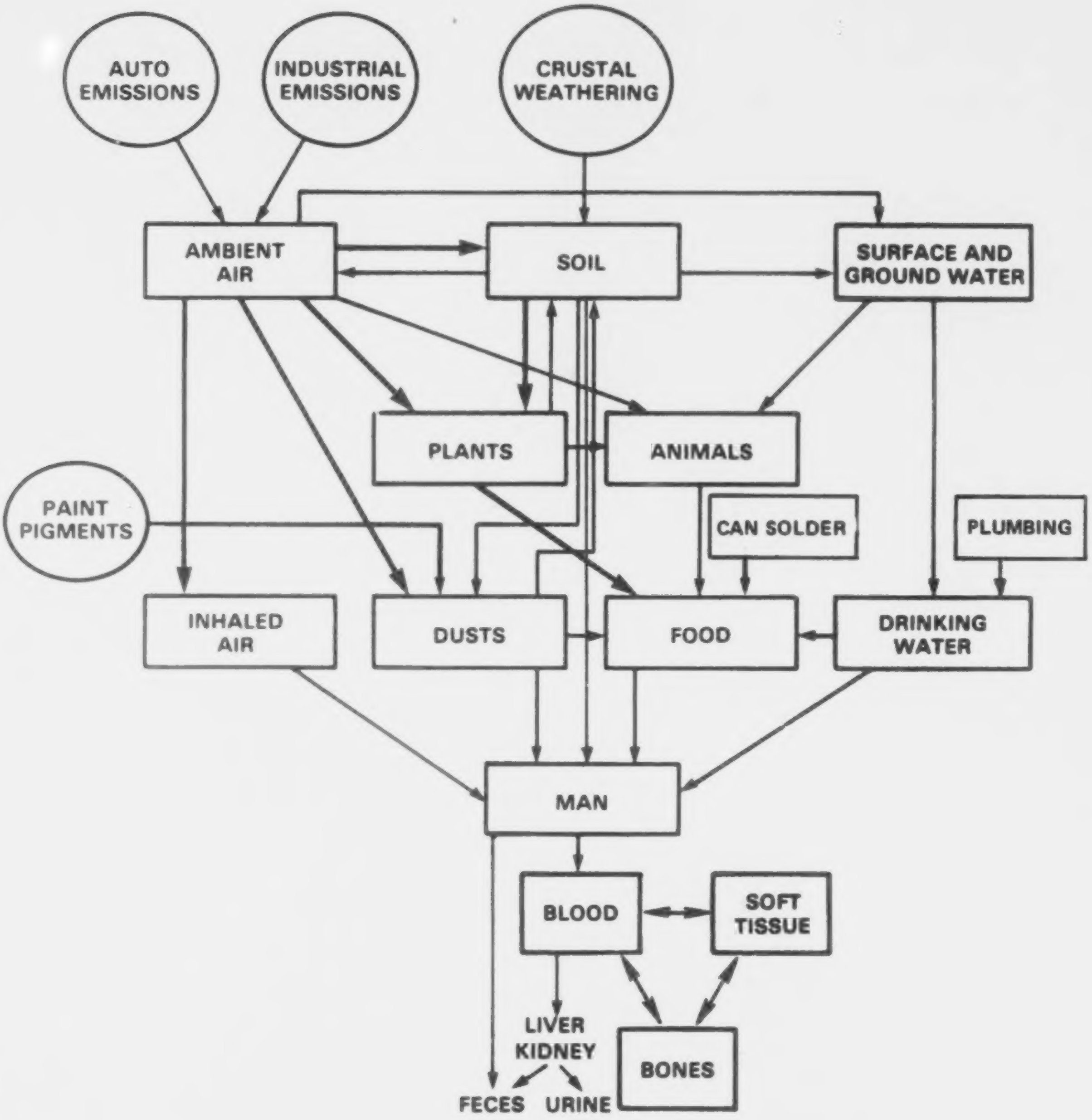
A. INTRODUCTION

Of the known environmental pollutants, lead has few rivals as a persistent cause of major public health concern. As an element, lead is indivisible and persists indefinitely as a discrete toxic substance. Therefore, lead put into the environment by human activities accumulates, adding to the total amount already there.

In the earth's crust, lead is present at only very low background levels. For centuries, however, extensive use has dispersed lead from geologic formations into many pathways of contact for human populations. Figure II-1 depicts various sources of lead and pathways or routes by which it biologically interacts with humans. The figure clearly shows that lead is now widely distributed in media that are pathways of intake/uptake for humans. Because of this ubiquitous dispersal, attempts at environmental reduction are difficult. To be fully successful, regulatory controls for lead should consider all pathways simultaneously.

One indication of the pervasiveness of lead contamination was provided by Patterson (1965), who calculated that the blood lead level of early, pre-industrial humans was 0.5 μg/dl, or about 15 to 30 times lower than the current levels seen in some segments of the U.S. population. Piomelli et al. (1980) measured blood lead levels in a remote population of the Himalayas and found an average Pb-B level of 3.4 μg/dl, which is 3- to 5-fold lower than the levels in parts of the U.S. population.

As noted later in this report, lead causes a broad range of adverse health effects in humans and experimental animals because it interferes with the normal functions of cells in general and calcium pathways in particular. These



AUTO EMISSIONS
INDUSTRIAL EMISSIONS
CRUSTAL WEATHERING
AMBIENT AIR
SOIL
SURFACE AND GROUND WATER
PLANTS
ANIMALS
PAINT PIGMENTS
CAN SOLDER
PLUMBING
INHALED AIR
DUSTS
FOOD
DRINKING WATER
MAN
BLOOD
SOFT TISSUE
LIVER
KIDNEY
BONES
FECES
URINE

Figure II-1. Pathways of lead from the environment to man and body disposition of lead.

Source: Adapted from EPA (1986a).

health effects span the human toxicologic spectrum, from classically known clinical effects in the nervous, red blood cell, and kidney systems, to relatively subtle biological effects in these and other systems that signal the onset of increasingly severe outcomes. Some of these effects have only recently been discovered with the advent of more sophisticated research techniques. In many cases, however, knowledge of the adverse effects of lead on humans dates back to the Greco-Roman era and beyond (see, e.g., Wedeen, 1984). Given this historical perspective, it is perhaps surprising that public health concerns did not earlier accelerate efforts to limit the amount of lead entering the U.S. environment or, at a minimum, prompt more debate over the acceptable trade-off between economic utility and adverse health effects.

The use of lead increased greatly in the 19th and 20th centuries; it was used in new ways and more of it was used in old ways. In at least one case, the commercial advantages of lead may have specifically maximized the probability of an adverse health impact. Paint high in lead content had the advantage of continuous chalking inside of homes and weathering outside of homes, thereby providing renewed surfaces and a fresh look longer. At the same time, however, dispersion of lead into the environment occurred.

The evolution and recognition of childhood lead poisoning as a public health problem in the United States and elsewhere has been described in detail by Lin-Fu (1982a). This problem is understood to encompass *in utero* lead intoxication as well. In the 18th and 19th centuries, sterility, abortion, stillbirth, and premature delivery were recognized among female lead workers and the wives of lead workers. Mortality was high in their offspring, as was the incidence of low birth weight, convulsions, failure to thrive, and mental retardation. With progress in industrial hygiene, the number of overtly lead-intoxicated workers was significantly reduced and concomitant reductions in the incidence of severe reproductive effects became apparent over time (U.S. EPA, 1986a).

The prevalence of direct lead poisoning in children was first examined in Australia in the 1890s (Gibson et al., 1892; Gibson, 1904), and the poisoning was traced to lead-based paint used on exterior railings and walls. In the United States, skepticism among physicians about lead poisoning in children did not lessen until Blackfan (1917) reported lead as the source of acute encephalopathy in a number of children. Physicians eventually recognized lead poisoning in children as a clinical entity (McKhann, 1926).

In the 1930s and 1940s, and accelerating through the 1950s and 1960s, the epidemiologic data base describing childhood lead poisoning rapidly expanded. In the 1950s and 1960s, even rudimentary efforts to screen numbers of children showed clear evidence of excessive lead exposure, at alarmingly high prevalence rates, among inner-city children.

A better appreciation of the U.S. lead problem started to emerge in the late 1960s and early 1970s, when Congress enacted the 1970 Clean Air Act and the 1971 Lead-Based Paint Poisoning Prevention Act, and the National Institutes of Health (NIH) and other public agencies began to fund significant research on human and experimental animal lead toxicology. The 1971 Lead-Based Paint Poisoning Prevention Act provided assistance to communities for lead screening and treatment programs, particularly the mass screening of children. Screenings began in mid-1971, after which childhood lead poisoning began to be recognized as a widespread public health problem. It came to be seen as a disease that could affect middle- and upper-class children, children living in rural and suburban areas, and those in low-income, inner-city families. Concurrently, exposure sources were often scrutinized and, as a result, the multimedia nature of the lead problem began to be identified.

The 1970 Clean Air Act established the U.S. Environmental Protection Agency (EPA). Through court action in 1975, EPA was required to evaluate atmospheric lead as a "criteria pollutant," in the terminology of the Clean Air Act. Since airborne lead readily enters other environmental compartments, EPA's assessment of lead as a criteria pollutant also began to define the problem in all segments of the human environment.

In the late 1960s, the NIH and other components of the U.S. Public Health Service began to fund comprehensive research on many aspects of the lead problem. Increasingly impressive evidence of the toxicologic potency of lead in humans and experimental animals was produced in the ensuing years. Results have ranged from documented excessive lead body burdens in industrialized countries (Piomelli et al., 1980) to those that show the subcellular mechanisms of lead toxicity (see, e.g., U.S. EPA, 1986a; Silbergeld, 1983a,b; and Silbergeld et al., 1980, for a comprehensive discussion).

To illustrate the U.S. lead problem, consider two categories of potential exposure: lead-based paint and the combustion of leaded gasoline. Pope (1986) estimated that in 1980 about 52% of all occupied residential units contained painted surfaces with lead at or above 0.7 mg lead/cm^2, a level judged hazardous to young children (CDC, 1985) and the lowest that can be reliably measured by field testing in situ. Even lower levels may be toxic. The above percentage equals about 42 million units, including some 21 million units built before 1940, which have the most leaded paint and are likely to be in the worst state of repair. These older houses are, therefore, the most probable source of lead exposure for young children. Lin-Fu (1982b) estimated that there are about 27 million pre-1940 units in the United States. Pope (1986) estimated, based on these and other data for pre-1940 housing, that 99% of all such housing had painted surfaces containing lead.

Leaded paint continued to be used in considerable amounts from 1940 until 1959. An estimated 70%, or 16 million housing units built during this interval, have painted surfaces that contain lead at levels above 0.7 mg lead/cm^2, based on Pope's best estimates from the survey data of Billick and Gray (1978), Lin-Fu (1985a,b), Schier and Hall (1977), the Arizona Department of Health Services (1976), and Gilsinn (1972). For the most recent housing units likely to have some leaded paint--those built between 1960 and 1974--Pope estimated on the basis of four surveys, that 20%, about 5 million units, have surfaces with lead levels exceeding 0.7 mg/cm^2.

Regarding the environmental magnitude of the lead-based paint problem in terms of mass, Clark, of the University of Cincinnati, OH, has estimated the total amount of leaded paint remaining in the 27 million or more housing units built before 1940 and the millions more built since 1940 (communication of J.J. Chisolm, Jr., M.D., to ATSDR, September 4, 1987). Annual U.S. lead use for white paint was identified for each year since 1910, drawing on relevant annual statistics of the Minerals Yearbook, U.S. Bureau of Mines. Since 1910, about 4.2 million short tons (3.9 million metric tons) of lead were used for white paint in the United States. Clark also estimated lead consumption for paint since the 1880s at about 7.0 million short tons (6.4 million metric tons).

These figures, as current environmental burdens, overestimate the extent of white paint lead in view of reductions through, for example, the demolition and remodeling of old houses. On the other hand, they significantly underestimate total lead paint production, because green, yellow, and black paints also were high in lead content but are not counted here. Overall, about 3 million tons of lead probably remain in paint accessible to children. This illustrative example takes on further meaning when one examines the millions of young children living in housing units having this lead burden (see Chapter V).

Regarding the combustion of leaded gasoline in the United States, from 1975 to 1984 a total of 1,087,800 metric tons of lead were consumed (U.S. EPA, 1986a). Virtually all of this lead was dissipated to the atmosphere and other areas of the environment. If lead emissions from earlier periods are included by assuming, in view of its long environmental half-life, that all of the dissipated amount accumulates, about 4 to 5 million metric tons of lead from gasoline remain in dust, soil, and sediments (U.S. EPA, 1986a).

When examining these figures, one also must consider relative dispersion factors that affect the amount of lead young children take in. The total amount of lead in paint may be roughly similar to that emitted from combusted gasoline, but the relative concentration of lead in media affected by the paint is significantly greater. Consider the amount of lead in paint chips or dust from weathered or chalked paint and the amount in dust and soil from traffic emissions: a 1% lead-paint film contains 10,000 parts per million (ppm) of lead, which is considerably higher than typical levels in dust or soil.

This report will document that lead is a pervasive environmental contaminant that causes a wide variety of adverse effects in humans. In short, lead is potentially toxic wherever it is found, and it is found everywhere.

B. DISCUSSION OF TERMS AND ISSUES

Some of the relevant terms used in Section 118(f) of the Superfund Amendment and Reauthorization Act (SARA) of 1986 that cover issues and concepts central to the childhood lead problem are defined and discussed in this section. These include:

(1) identification of those U.S. child population groups at greatest risk for lead exposure;
(2) types of exposure assessments that relate to quantifying lead exposure of individuals and populations;
(3) the most relevant meanings of the term "environmental lead sources," both for their specific identification and for the discussion of interrelationships among exposure pathways; and
(4) the nature of adverse health effects associated with lead exposure in human populations.

1. Young Children and Other Groups at Greatest Risk for Lead Exposure and Adverse Health Effects

Young children, particularly preschool children, are the subset of the U.S. population most at risk for excessive exposure to lead and its associated adverse health effects. The age interval for children at greatest risk has not been precisely defined. Although infants and toddlers are at particular risk because of their behavior patterns (e.g., hand to mouth activities) and other factors, various effects have been found in children up to 6 to 8 years of age. Since childhood lead exposure actually begins prenatally, i.e., during *in utero* development, and the effects of such exposure have been reliably measured in terms of both fetal and postnatal developmental impairments, an adequate assessment of the childhood lead problem must encompass the human fetus and its exposure via the pregnant woman.

Young children are vulnerable to the effects of lead for at least two reasons: their developmental physiology and their contact with parts of their environment contaminated by lead. Children differ from adults both qualitatively and quantitatively in their metabolism of lead. The Centers for Disease Control (1985) and U.S. EPA (1986a) have discussed these differences in detail, and this report provides a comprehensive summary of the physiological distinctions in Chapter III.

The evidence for identifying fetuses as a risk group is described in Chapters III and IV. As noted earlier, the congenital poisoning of children of lead workers in the 19th century was frequently severe and obvious, including often fatal outcomes (U.S. EPA, 1986a). Recent epidemiologic studies show that developmental toxicity also occurs at much lower levels of fetal lead exposure. These effects include impairments in postnatal neurobehavioral development up to two years of age, reductions in birth weight and gestational age, and possibly other effects at exposure levels that were prevalent and generally considered safe only a few years ago (Davis and Svendsgaard, 1987). Although these effects have been most clearly linked with prenatal lead exposure indices, the potential role of lead-induced gametotoxic effects on the egg and sperm may also be important (U.S. EPA, 1986a).

2. Monitoring Lead Exposure in Young Children and Other Risk Populations

Health professionals determine the degree of lead exposure of children and other risk populations in three ways. The traditional method is to measure lead in external or environmental media (ambient air, workplace air, food, water, dust, soil, etc.) that are pathways for human exposure. Surveying lead in these media, termed environmental or ambient monitoring, provides information about the lead exposure sources and the potential risk for persons so exposed to lead. Environmental monitoring does not indicate the actual internal level of lead for any given individual, because individuals may respond differently to an external source of lead exposure. On a group basis, however, one can determine the probable internal exposure level, given knowledge of the concentrations in the external media and the mathematical relationship between the two variables (different levels in the media and different responses in groups of people). In many cases, one must use environmental monitoring, either because other types of exposure assessment cannot be carried out or because a more direct, systemic measure of exposure is considered unnecessary.

Lead exposure is also commonly assessed through biological monitoring and effects monitoring. Biological monitoring is the measurement of the concentration of lead in a biological sample, e.g., blood, from an exposed person. Effects monitoring involves measuring some endpoint, e.g., the amount of certain proteins or enzymes associated with lead exposure. For a more comprehensive examination of monitoring exposure to lead, see Elinder et al. (1987). Biological monitoring and effects monitoring have advantages for assessing health effects, giving an integrated picture of a person's uptake of lead from all external sources.

To assess lead exposure in children and other risk groups, the level of lead in whole blood (Pb-B) is the most practical biological measure of ongoing lead absorption. This measure is used throughout Chapters IV-VIII as a means of relating lead exposure levels to adverse health effects. A Pb-B level is generally regarded as reflecting relatively recent exposure. However, Pb-B levels also give information on the relative level of exposure at more remote time points. In other words, a child who had the highest Pb-B level at one time will probably have the highest Pb-B level at a future retesting. Pb-B levels do not, however, indicate cumulative past exposure, as do lead levels of mineralizing tissue such as tooth or bone. Such cumulative measures cannot yet be routinely employed in screening programs. The kinetic, toxicological, and practical aspects of biological monitoring and other approaches to assessing lead exposure have been extensively discussed by U.S. EPA (1986a).

In young children, effects monitoring for lead exposure is primarily based on lead's impact on the heme biosynthetic pathway, as shown by (1) changes in the activity of key enzymes delta-aminolevulinic acid dehydratase (ALA-D) and delta-aminolevulinic acid synthetase (ALA-S), (2) the accumulation of coproporphyrin in urine (CP-U), and (3) the accumulation of protoporphyrin in erythrocytes (EP). For methodological and practical reasons, the EP measure is the effect index most often used in screening children and other population groups.

Effects monitoring for exposure in general and lead exposure in particular has drawbacks (Friberg, 1985). Effects monitoring is most useful when the endpoint being measured is specific to lead and sensitive to low levels of lead. Since EP levels can be elevated by iron deficiency, which is common in young children, indexing one relationship requires quantitatively adjusting for the other.

An elevated Pb-B level and, consequently, increased lead absorption may exist even when the EP value is within normal limits, now ≤35 micrograms (μg) EP/deciliter (dl) of whole blood. We might expect that in high-risk, low socioeconomic status (SES), nutrient (including iron)-deficient children in urban areas, chronic Pb-B elevation would invariably accompany persistent EP elevation. Analysis of data from the second National Health and Nutrition Examination Survey (NHANES II) by Mahaffey and Annest (1986) indicates that Pb-B levels in children can be elevated even when EP levels are normal. Of 118 children with Pb-B levels above 30 μg/dl (the CDC criterion level at the time of NHANES II), 47% had EP levels at or below 30 μg/dl, and 58% (Annest and Mahaffey, 1984) had EP levels less than the current EP cutoff value of 35 μg/dl (CDC, 1985). This means that reliance on EP level for initial screening can result in a significant incidence of false negatives or failures to detect toxic Pb-B levels. This finding has important implications for the interpretation of screening data, as discussed in Chapter V.

3. Environmental Sources of Lead in the United States with Reference to Young Children and Other Risk Groups

As graphically depicted in Figure II-1, several environmental sources of lead exposure pose a risk for young children and fetuses. Many sources not only contribute to direct or proximate exposure but also to indirect exposure via secondary processes. Pathways for human exposure to lead include paint, dust and soil, drinking water, air, and food. Many of these pathways contribute indirectly to exposure via other media. For example, airborne lead fallout contributes to dust and soil lead levels, which are also increased by chalking and weathering of lead paint, both on the interior and exterior of structures.

Mobile and stationary sources of lead are referred to in this report. Mobile sources refer to automobiles and other vehicles that burn leaded gasoline; stationary sources encompass a constellation of lead production, processing, and end-use operations, including primary lead smelters and refineries, secondary lead smelters, and municipal incinerators (U.S. EPA, 1986a). All of these sources emit lead to the atmosphere.

Environmental sources of lead differ in their quantitative impact on a given human population, in their geographic distribution, in their relative persistence as a long-term exposure source, and in the types of problems they pose for abatement strategies. For example, sources of high-level lead exposure for children in older population centers are lead in paint and lead in dust and soil, particularly in areas of deteriorated housing and heavy traffic. In rural areas, remote from heavy vehicular traffic but near stationary lead operations, children with no leaded paint exposure can still be significantly exposed by inhaling airborne lead particles emitted directly from a facility and by inhaling or ingesting lead in dust inside and outside the house and in contaminated soils (U.S. EPA, 1986a). In some agricultural areas, prior use of lead arsenate as a pesticide may pose a lingering exposure risk, but a risk that is poorly understood. In new housing units, children not otherwise in contact with lead can be exposed to high levels in their drinking water because of the lead in plumbing solder, which is of particular concern where the water is naturally corrosive. Sources are discussed in detail in Chapter VI, which gives estimates of children exposed by source category.

Lead is a multimedia pollutant. Therefore, one cannot speak, in biological terms, about lead exposure by isolated sources of contact. The physicochemical and toxicokinetic properties of lead produce a net body burden or target organ burden in humans, and it is this net burden that is related to the risk of adverse health effects.

In discussing the legislative history of multimedia pollutants, Mushak and Schroeder (1981) pointed out that because of legislative mandates various agencies have traditionally carried out their activities on a medium-by-medium basis, with, in many cases, little coordination. Specific laws have called for the assessment and control of the hazards posed by separate media. These laws have created separate standard-setting procedures and criteria, almost always administered by separate agencies or divisions within agencies. Mushak and Schroeder (1981) also pointed out that the Environmental Law Institute identified 23 statutes affecting toxic agents (such as lead), most of which are found in many environmental media but which have been approached by regimens for a single medium. Lately, however, more coordinated efforts have been used in dealing with multimedia problems. Recent legislation calling for more interagency coordination has bolstered this move.

Given the complexity of lead exposure in children, the directive in Section 118(f) to rank children according to exposure source must be interpreted to mean ranking by the major environmental source. Other factors that interact with lead exposure must also be considered. Finally, it should be recognized that even though no single source of exposure may be dominant, the aggregate absorption of lead from many sources may produce unacceptable internal lead levels. These points are discussed further in Chapter VIII.

4. Adverse Health Effects of Lead in Young Children and Other Risk Groups and Their Role in Health Risk Assessment

Lead exposure in young children is associated with a broad range of toxicologic effects, such effects being dose-dependent and inducible at relatively low exposure levels. These adverse health effects are discussed in detail in EPA's Air Quality Criteria for Lead (U.S. EPA, 1986a) and in the CDC's most recent statement on lead poisoning in children (CDC, 1985). Historically, the definition of lead poisoning has progressively expanded in terms of the number of significant effects and the lowest levels of lead exposure associated with these effects.

As discussed by U.S. EPA (1986a), exposure to lead produces a continuum of toxic responses. Definitive criteria for judging which lead-related effects are adverse are not easy to state, but some are now accepted as reasonable for judging lead-associated lesions in humans. These include (1) impaired functioning of a tissue or organ system; (2) reduced reserve capacity of tissues and organs to deal with stresses induced by other toxicants or xenobiotics; (3) the reversibility or irreversibility of the effect; (4) the relative frequency of an effect in a population; (5) the presence of the effect in that population (e.g., young children), most vulnerable to the toxicant; and (6) the cumulative or aggregate impact of various effects within single organ systems on the well-being of the entire organism.

Impacts of lead on target organs and systems are evident across a broad exposure range, and effects at low exposures have only more recently been judged to meet the criterion of health impairment (see, e.g., CDC, 1985). An illustration of this point is the traditional term "anemia" when applied to lead poisoning. As a measure of lead effect, the term has come to mean a cluster of adverse effects on heme biosynthesis and erythrocyte stability. These effects are seen at lower levels of lead exposure than have been classically defined as lead anemia. Implicit in accepting these early effects as significant is the notion that preventing such outcomes prevents more serious responses. In other words, preventive medicine has a role in dealing with the effects of exposure to lead.

Lead-induced impairments of the body's capacity to offset various stresses from other agents appear to result from reduced hepatic metabolism, even at moderately elevated Pb-B levels (Saenger et al., 1984). As noted by U.S. EPA (1986a), a number of studies show that the body is less able to detoxify foreign substances such as drugs and toxic pollutants because lead reduces hepatic heme, upon which enzyme biosynthesis depends.

An important criterion for assessing the significance of lead-induced effects is whether an effect is reversible or irreversible, usually meaning a physiological return to normal. In addition, there is the important public health question of whether exposure conditions will be reversible. As noted by EPA (1986a), if removing a subject from lead exposure is highly unlikely for socioeconomic or other reasons, then adverse effects, reversible or not, will persist and operate as *de facto* irreversible effects. For example, effects of lead on heme biosynthesis in children can technically be reversed by removing lead exposure. When these effects occur in inner-city, low-SES children who remain in a high-risk setting indefinitely, effects persist during the period of their highest vulnerability (see, e.g., CDC, 1985, and EPA, 1986a).

The issue of aggregate impact on the overall well-being of humans because of effects on single systems is important in assessing lead toxicity. As discussed in Chapter IV, a disturbance in one system or at one stage of development may have far-reaching ramifications for other systems or later stages of development. Such cascading effects have been well described for the multi-organ impacts of heme reduction (see Figure IV-2).

III. LEAD METABOLISM AND ITS RELATIONSHIP TO LEAD EXPOSURE AND ADVERSE EFFECTS OF LEAD

Lead "metabolism" (pharmacokinetics, toxicokinetics, biokinetics) refers to the various integrated human body processes that govern the intake, absorption, distribution, and retention/excretion of lead. These processes may be basically kinetic or they may describe interactive relationships that affect the biokinetic behavior of lead. These elements of in vivo behavior form the central link between the relatively simple event of external exposure to lead and the ultimate complex manifestation of some lead-associated effect or risk for the effect. It is from this particular perspective that a brief discussion of the topic is provided for the benefit of the general reader.

Some points of interest include: (1) those characteristics of lead's metabolic behavior that abet its status as a public health issue; (2) the role of lead metabolism in defining certain subsets of the general population as being at special risk for the adverse effects of lead; and (3) the underlying metabolic characteristics of lead that affect the use of various biological exposure indicators that are widely employed in later chapters of this report.

A. LEAD ABSORPTION IN HUMAN POPULATIONS

Lead enters the bloodstream from several body compartments, with the rate of absorption (uptake) depending upon the chemical and physical forms of lead and physiological characteristics, for example, nutritional status and age, of the populations exposed to it.

1. Respiratory Absorption of Lead in Human Populations

Lead in inhaled air is absorbed eventually in a two-part process: some fraction of the inhaled air lead is deposited in the pulmonary and higher parts of the respiratory tract, and some amount of this deposited lead burden is

111

absorbed, either in situ in the case of the pulmonary compartment or via retrociliary movement to the buccal cavity and swallowing. In the latter case, absorption can occur from the gastrointestinal tract.

In adult humans exposed to lead in ambient air only, the pulmonary deposition rate is 30 to 50% (see U.S. EPA, 1986a; Chamberlain, 1983; Morrow et al., 1980; Chamberlain et al., 1978). Essentially all of this deposited amount is absorbed over a relatively short time (Chamberlain et al., 1978; Morrow et al., 1980; and Rabinowitz et al., 1977).

The respiratory absorption rate is relatively fixed over a broad range of air lead concentrations that nonoccupational populations are likely to encounter, and the rate is generally the same for a number of chemical forms of lead e.g., chloride and oxide (Morrow et al., 1980; and Chamberlain et al., 1978). Breathing rate changes and the particle size of lead-containing particulates in air also affect the absorption rate, but the percentages given above refer to typical exposures of general populations.

Most of the available quantitative information has been derived from adults, and comparisons with children under conditions of identical exposure to airborne lead are difficult with respect to relative respiratory dynamics. Young children inhale a proportionately higher daily air volume per unit measure (weight, body area) than do adults (Barltrop, 1972). This is related to higher metabolic demand and therefore higher gas exchange rate. James (1978) has estimated that children have a lung deposition rate of lead that can be up to 2.7-fold higher than in adults on a unit body mass basis. This estimate is supported by anatomical modelling approaches for airway lengths as a function of age and development (Hofmann et al., 1979). The data of Hofmann et al. (1979) indicate that there is a period of maximum childhood intake, specifically at about 6 years of age.

2. Gastrointestinal (GI) Absorption of Lead in Human Populations

In nonoccupationally exposed populations, the lead absorbed by the GI tract is from the intake of lead in foods and beverages in the case of adults and older children and from the intake of both foods and nonfood items, for example, lead-contaminated dusts and soils, in preschool children. Young children take in nonfood lead via normal mouthing activity and other behaviors associated with oral exploration of the environment. An extreme manifestation of this otherwise common behavior is pica, a behavioral trait associated with particularly severe lead poisoning. This topic is addressed in detail elsewhere (U.S. EPA, 1986a; National Research Council, 1976, 1980).

In the adult human, the rate of GI lead absorption from typical diets is 10 to 15% of the ingested quantity (Gross, 1981; Rabinowitz et al., 1980; and Chamberlain et al., 1978). This rate range applies to enteric lead assimilated during usual meal times. Under fasting conditions, such as imbibing lead-containing beverages between meals, this rate can increase dramatically (Heard and Chamberlain, 1982; Rabinowitz et al., 1980; and Blake, 1976). The amount can increase to 60% or more absorption (Heard and Chamberlain, 1982). A major factor in this increase during fasting is the lower amount of those dietary components that are known to suppress uptake and that are present when meals are ingested. These components are discussed in more detail later in this chapter (Section D).

The GI lead absorption rate in infants and children, about 50%, is considerably greater than the rate in adults (Ziegler et al., 1978; and Alexander et al., 1973) indicating a higher relative exposure rate for a given food intake. It is possible that in the Alexander et al. (1973) study, enhanced absorption was influenced by iron deficiency, which is common in that group. However, the Ziegler et al., 1978 study was not complicated by this factor because of the age interval of the children.

Related to age-dependent lead absorption from the human GI tract are data from the second National Health and Nutrition Examination Survey (NHANES II) (Mahaffey et al., 1982), which show a moderate peaking in Pb-B at about 18 to 24 months of age. This peak may reflect intrinsic differences in enteric uptake rates at different ages, the highest propensity for ingesting lead-contaminated nonfood items at this age, or both. With regard to the latter factor, estimates of the daily amount of dust or soil young children ingest vary (see Chapters 10 and 11, U.S. EPA, 1986a), but recent empirical studies from a smelter community (Centers for Disease Control, 1986a) yield a range of 121 to 184 mg with one marker technique, and an upper determination of 1,834 mg--about tenfold higher--with another technique. Whatever figure is selected, soil and dust of even moderate lead content clearly can contribute markedly to the oral lead intake.

The question of dietary lead bioavailability as a function of such properties as either the chemical or physical form of lead or its matrix (for example, meat or beverages) has been addressed in various studies. In this context, bioavailability refers to the actual quantity being absorbed, rather than the total amount in the gut. Heard and Chamberlain (1982) and Rabinowitz et al. (1980) noted that various chemical forms of lead were equally absorbed and that the dietary matrix imparted a minimal effect. A useful study, albeit one with experimental animals, is that of Dacre and Ter Haar (1977). Results show that lead in soils and road dusts from air lead fallout is absorbed to the same extent in the rodent GI tract as is lead in simple water solutions. This indicates that lead in such material is potentially highly bioavailable when young children ingest it in the course of normal behavior.

Within the range of lead levels in food likely to be encountered by even heavily exposed populations, the absorption rate from this route remains the same (Heard and Chamberlain, 1983; Flanagan et al., 1982; and Blake, 1980). Flanagan and co-workers (1982), for example, observed the same absorption rate for a lead dose of 400 μg as for 4 μg.

3. Percutaneous Absorption of Lead in Human Populations

The human body absorbs very little lead through the skin, when considering the inorganic ion of lead (the form of interest). Results of one detailed study (Moore et al., 1980) showed that the human skin absorbs an average of about 0.06% of the applied quantity.

4. Transplacental Transfer and Fetal Uptake of Lead in Pregnant Women

For the fetus, the route of lead uptake is across the placenta. Lead readily crosses the placental barrier during the entire gestation period, including that critical period when the nervous system is being embryologically formed. Fetal lead uptake is cumulative until birth (Rabinowitz and Needleman, 1982; Alexander and Delves, 1981; and Barltrop, 1969).

B. DISTRIBUTION OF ABSORBED LEAD IN THE HUMAN BODY

For the issues addressed in this report, the *in vivo* lead is distributed to two types of receiving tissues: soft tissues, including organs considered targets of lead's toxic action, and mineralizing systems, such as teeth and bone. Bone is not only affected toxicologically by lead but also serves as the body's major storage site. Such a tissue as bone accumulates lead at a significant rate for much of the human life span and poses a risk as a potential source of mobilizable, endogenous toxicant.

With respect to the biokinetics of lead movement and models of systemic behavior, lead in the human body can be viewed as being distributed to at least three compartments (see, e.g., Rabinowitz et al., 1976). Lead in the blood is in its most labile form; lead in soft tissue is somewhat more stable; and lead in bone accumulates steadily in several subcompartments, which differ in allowing lead mobility back to blood.

Absorbed lead enters the bloodstream, where, under fairly steady exposure conditions, at least 99% becomes bound to the erythrocytes (DeSilva, 1981; and Everson and Patterson, 1980). With constant exposure, movement of lead from blood to tissues and back to blood establishes a near equilibrium. In short-term experimental studies for adults, lead movement from blood has a half-life of about 25 days (Chamberlain et al., 1978; and Rabinowitz et al., 1976). Pb-B levels will rise or decline, depending on the direction of the exposure change.

For adults with abruptly increased exposure, e.g., new lead workers, Pb-B increases to a new "steady-state" level after about 60 days (Griffin et al., 1975; and Tola et al., 1973). With a marked decrease in exposure, the level to which Pb-B declines and the time required to reach this new level are a complex function of the existing body lead burden, the specific nature of the study design, and the total time of prior exposure (Hryhorczuk et al., 1985; Kang et al., 1983; and O'Flaherty et al., 1982). When lead workers are removed from exposure in the workplace, Pb-B levels may not fully decline for months or years.

Changes in Pb-B of children in the course of development and because of changes in exposure may occur over a slower time frame. Succop et al. (1987) recently reported that the biological half-life of Pb-B of 2-year-old children is about 10 months.

1. Lead Uptake in Soft Tissues

In human soft tissue, including the various target organs for toxic action, lead uptake occurs in a complex fashion that reflects the specific tissue kinetics, the external exposure level, the lead level in circulating blood, and other factors. With few exceptions, levels in soft tissue range from about 200 to 500 parts per billion (ppb), and the levels stabilize in nonoccupationally exposed populations by early adulthood (see, for example, Barry, 1981 and 1975). The 200- to 500-ppb levels of lead for tissues in unexposed populations need not rise very much to indicate toxicity as we index its signs and symptoms today. For example, the lead content in human brain tissue with severe acute or chronic neurotoxic damage is, in many cases, only 1 to 2 ppm or even less, demonstrating that lead can be a potent toxicant at very low levels in tissues. As a second example, Pb-B levels approximating 100 to 150 ppb (10 to 15 μg/dl) are associated with early toxic effects in human tissues now recognized as targets of toxic action.

In general, lead does not appear to accumulate in soft tissues with age, and this includes the lung. Such nonaccumulation in the lung is further evidence that lead is quickly absorbed from the respiratory tract, as discussed earlier. Some evidence suggests that lead accumulates in human brain tissue with heavy exposure encountered in some occupational settings (Barry, 1975). The question of whether lead accumulates with age in soft tissues is still undecided on statistical grounds, since the usual cross-sectional analysis approach of using pooled autopsy samples from subjects of varying age would obscure small but toxicologically important accumulative changes in human populations. This contrasts with lead in mineralizing tissues, where the accumulation rate with age is so huge that the mode of analysis would not obscure it.

The central and peripheral nervous systems of humans, especially key brain regions vulnerable to lead, have no apparent barrier to lead uptake, and this uptake appears to differ among brain regions. Consequently, increased exposure translates to increased entry of lead into the brain across the total exposure range.

2. Lead Uptake in Mineralizing Tissue

Many studies, employing both biopsy and autopsy data, have documented that lead primarily localizes in mineral tissue such as bone and teeth and that it continuously accumulates in these sites for most of the human life span (Barry, 1975, 1981; U.S. EPA, 1986a; National Research Council, 1980). Such uptake apparently begins *in utero* (Barltrop, 1969) and occurs across all ranges of exposure--that is, there is no threshold for lead uptake in bone.

The total body lead content can approximate 200 mg and more in the absence of occupational exposure and can rise to well above this in lead workers. Of the total body content, at least 95% is lodged in mineral tissue (Barry, 1975; Rabinowitz et al., 1976).

Most of the lead in bone had long been considered to be metabolically inert, that is, nontoxic. However, bone now is recognized as a living organ that is sensitive to the toxic effects of lead (Rosen, 1983, 1985); furthermore, a fraction of its total lead content is more mobile than it is often assumed to be (U.S. EPA, 1986a). This mobile fraction can be resorbed into blood and potentially can exert toxic effects, as discerned from kinetic modelling (Rabinowitz et al., 1976), chelation mobilization (Saenger et al., 1982; Piomelli et al., 1984; Araki and Ushio, 1982), and experimental animal studies (e.g., Hammond, 1973).

Lead also accumulates in teeth as a complex function of age (Steenhout and Pourtois, 1981), tooth region, e.g., enamel, secondary dentine, etc. (Needleman and Shapiro, 1974), and tooth type (Delves et al., 1982). Lead accumulates especially in the dentine of childrens' teeth and continues to accumulate until the teeth are shed. Various researchers have used this fact to index cumulative lead exposure in children (see, e.g., Needleman et al., 1979).

C. LEAD EXCRETION AND RETENTION IN HUMAN POPULATIONS

Absorbed lead in the human body that is not retained is eliminated in either urine (about 65%) or bile (35%). The available evidence indicates that about 50% to 60% of daily absorbed lead is rapidly excreted (Chamberlain et al., 1978; and Rabinowitz et al., 1976), with the balance being distributed to bone. This rapidly excreted fraction has a half-life in the body of about

3 weeks (Chamberlain et al., 1978; Rabinowitz et al., 1976), which approximates the half-life of lead's movement from blood. Of the fraction of lead moved to bone, about half or 25% of the amount originally absorbed will soon be resorbed into blood and then excreted.

The rate at which the body eliminates lead depends on age, with young children excreting less of a daily uptake than adults because of greater net movement of lead to bone. This comparative behavior is depicted in Table III-1, in which the data of Ziegler et al. (1978) for infants are compared with data from two adult groups for which information was complete enough for the necessary calculations.

TABLE III-1. COMPARATIVE DIETARY LEAD METABOLISM IN INFANTS AND ADULTS[a]

Variable	Infants[b]	Adults[b]	
		Group A	Group B
Dietary intake	10.8	3.6	3.9
Fraction absorbed	0.46 (0.55)[c]	0.15	0.15
Dietary Pb absorbed	5.0 (5.9)	0.54	0.58
Air Pb absorbed	0.20	0.21	0.11
Total absorbed Pb	5.5 (6.1)	0.75	0.68
Urinary Pb excreted	1.00	0.47	0.34
Ratio: urinary/absorbed Pb	0.19 (0.16)	0.62	0.50
Endogenous fecal Pb	0.5 (1.6)	0.24	0.17
Total excreted Pb	1.5 (2.6)	0.71	0.51
Ratio: excreted/absorbed Pb	0.3 (0.4)	0.92	0.75
Fraction retained	0.34 (0.33)	0.01	0.04

[a] Adapted from U.S. EPA, 1986a, and references cited therein.

[b] μg/kg body weight.

[c] Corrected for endogenous fecal lead when values are in parentheses.

The body's lead excretion rate as a function of the amount of lead absorbed has not been well studied. Chamberlain (1983) examined a number of clinical and epidemiological studies and produced an aggregate analysis showing that as lead intake and uptake increases, so does the excretion rate.

Among retention rates for metal toxicants in humans, that for lead is relatively high, mainly because lead accumulates in bone and teeth. Lead in bone accumulates with age until about 60 years of age, when changes in diet and/or mineral homeostasis lead to a net negative balance; that is, daily loss exceeds the daily intake. However, beyond the years of active accumulation, little total body lead is lost. The half-life of lead in the most dense, mineral portion of bone is about 20 years or more for the active accumulation phase in humans.

Lead can be mobilized from some subcompartment within human bone during various physiological stresses, specifically pregnancy and lactation. Increases in Pb-B have been documented during pregnancy for a population of smelter region women (Zaric et al., 1987) and during lactation in the careful isotope tracing study of Manton (1985). Of equal importance, Manton (1985) showed that Pb-Bs at steady state also had sizable inputs from bone lead in two subjects.

In young children, the level of lead retention is considerably higher than in adults (Ziegler et al., 1978; Alexander et al., 1973), and the location of these higher deposits is unknown. A key factor in lead disposition in children is the metabolic fact that in the first decade of life the skeleton grows exponentially, increasing about 40-fold. Body growth overall and bone uptake of lead result in a huge dilution factor in lead concentration. A more meaningful measure here is the lead content of bone in children of various ages, which is discussed in detail in U.S. EPA, 1986a.

D. METABOLIC INTERACTIONS OF LEAD WITH NUTRIENTS AND OTHER ACTIVE FACTORS IN HUMAN POPULATIONS

The interplay of lead metabolism and the physiological status of humans, especially nutritional well-being, figures importantly in the level of lead exposure required to produce effects and manifestations of toxicity. A number of agents interact with lead entering the human body, and the available data consist of both human studies and experimental animal models of such interactive behavior. A detailed presentation of such interactions has been set forth in U.S. EPA (1986a), and only summary statements are provided here.

The major interacting agents of interest, also discussed in Chapter IX as part of exposure prevention strategies, are calcium, iron and, to some extent, phosphorus.

Mahaffey and co-workers (1976) reported that children with elevated Pb-B had lower intakes of calcium and phosphorus from diet than did a reference population. Similarly, Sorrell and co-workers (1977) found that blood lead was inversely correlated to dietary calcium intake at a high level of statistical significance. Similar results were reported by Johnson and Tenuta (1979) and

Ziegler et al. (1978). Experimental data gathered with human volunteers by Heard and Chamberlain (1982) show that lead absorption is significantly suppressed when lead is administered in diet with calcium and/or phosphorus.

Iron deficiency has long been known to be a key factor in the level of systemic lead exposure for young children, especially for those children from poor, inner-city families (Chisolm, 1981; Yip et al., 1981; Watson et al., 1980). A number of studies (e.g., Chisolm, 1981; Yip et al., 1981; Watson et al., 1980) have established a strong inverse correlation between iron status and Pb-B.

The main conclusion to be drawn from studies of lead-nutrient interactions is that defects in nutrition will enhance lead absorption/retention and toxicity risk. This problem is amplified in children where nutrient deficiencies are commonplace and where lead exposure is highest, i.e., 2- to 4-year-old, underdeveloped children.

Zinc deficiency can also play a role in enhanced lead absorption and toxicity risk (Markowitz and Rosen, 1981) and can be an interactive factor for children with zinc deficiency because of sickle cell anemia.

E. LEAD METABOLISM AND SOME KEY ASPECTS OF LEAD EXPOSURE AND TOXICITY

The metabolic behavior of lead in the human body has a major influence on the status of lead as a persisting U.S. public health problem. There are at least three points of connection: (1) lead metabolism as a factor in its behavior as an insidious toxicant even in populations with moderate exposure; (2) differential lead metabolism as a factor in identifying risk populations for both exposure and toxicity; and (3) lead metabolism as it relates to the accuracy and usefulness of biological indicators such as Pb-B in defining the toxicity risk for target tissues.

1. Lead Metabolism and the Nature of Lead Exposure and Toxicity

In general, the metabolic behavior of a toxicant is intimately related to toxic responses, which also pertains to lead. With lead, however, some points needing further discussion relate to its propensity to accumulate in the body over time and over a wide range of lead exposures in various intake media with no threshold for the phenomenon in terms of smallest intake rates.

When considering information showing that (1) lead can be mobilized from sequestration in bone back to the bloodstream, (2) such stresses as pregnancy and lactation may alter mineral homeostasis enough to effect such mobilization, and (3) low levels of circulating lead are associated with postnatal and prenatal effects in human populations, we can offer the following conclusions about lead as a unique public health problem:

(a) Since lead is a cumulative human toxicant, the usual dose-effect and dose-response relationships must be applied cautiously to lead in human populations (see Chapter IV). For example, when we speak of integrated lead exposure in a target tissue, that is, toxicity defined as a lead level over some time period, accumulated lead with a slow removal time may be inherently more toxic than lead moving rapidly in and out of tissues.

(b) The accumulation of lead in human body compartments necessitates attention to toxicity risks even if only low levels of lead are being absorbed at a given time. Low intake levels in each of many intake sources, for example, food and water (see Chapter VIII) further complicate this issue.

(c) The above problems of integrative exposure, cumulative dose-effect behavior, and public health consequences are even further complicated by new prospective studies that document numerous adverse effects in infants that can be traced to *in utero* exposure, that is, exposure to maternal lead during gestation. There is no metabolic barrier to fetal lead uptake. Furthermore, the amount of lead maternally circulating for fetal uptake may actually be higher than usual if the physiological stress of the pregnancy disrupts the metabolism of minerals in bone and releases lead stored there.

(d) The interplay between systemic lead exposure due to ongoing external exposure and that due to accumulated toxicant in bone and elsewhere has been examined. For example, Christoffersson and co-workers (1984) used *in vivo* measurement of bone lead to show that when human subjects are actively exposed to lead, the blood lead best correlates with the concurrent lead exposure. However, when active exposure stops, blood lead remains troublesomely elevated and is highly correlated statistically with the lead level in the bones of these subjects. These data show that the circulating blood lead is a function of both ongoing and cumulative past exposure (bone lead). The latter contributes via resorption from bone back to blood.

2. Lead Metabolism and the Identification of Risk Populations for Lead

Those occupationally exposed to lead are an obvious risk population, but one outside of the purpose of this report. At present, we can identify at least two risk populations for lead exposure and toxicity among the nonoccupationally exposed general population. These two risk groups from the general U.S. population are young children and pregnant women, the latter because of the exposure of the vulnerable fetus. Metabolic criteria define these subsets as risk subjects.

Young Children as a Risk Group on Metabolic Grounds

Young children absorb a greater percentage of ingested lead per unit body measure than do adults. They also take in more lead-containing dietary components per body unit measure because of caloric and energy demands. In addition to having a greater absorption rate, children retain a greater fraction of lead. This differential is distributed to a rapidly growing skeleton and to soft tissues. Growth dilutes the dose function, but the total lead in the skeleton shows marked net accumulation and can serve as an endogenous source of exposure. Children are most vulnerable to lead effects at the exact time during which they physiologically most need optimal nutritional status. Deficiencies in elements that otherwise suppress lead absorption and toxicity place poorer children at greater risk, since such children are most often poorly nourished.

Children are likely to undergo stresses such as chronic diseases that may affect mineral metabolism and lead release from bone. This possibility has not been examined in any detail in children, but we do know that physiological stress such as pregnancy and lactation will mobilize bone lead in women.

Furthermore, behavioral traits in young children interact with lead metabolism to define added risks for this group. Normal mouthing activity enhances lead intake and uptake, a subject pursued in more detail in Chapters VI and VIII. Lead bioavailability in nonfood categories (soil, dust, paint chips, etc.) is assumed to be rather high, and these sources contribute to body burden and toxicity risk.

The Fetus (Pregnant Women) as a Risk Category on Metabolic Grounds

As noted, there is no metabolic or anatomical barrier to lead for the fetus, and uptake continues for the entire gestational period, including the embryological development period for the nervous system and other targets of lead toxicity. The exposure of pregnant women is, therefore, of key importance, especially given the evidence of *in utero* effects at low lead levels (see Chapter IV).

3. Lead Metabolism and Biological Exposure Indicators

The usefulness of biological or body indicators of internal lead exposure is essentially determined by the metabolic behavior of the toxicant. From the foregoing discussion in this chapter, lead metabolism tells us that:

a. The actual level of lead in blood is a more labile measure and will reflect more recent exposure. However, Pb-B can give relative ranking in an exposure group over somewhat extended periods, which may reflect continuing lead washout from accumulating sites.
b. Accumulating tissues, such as bone and teeth, give a cumulative picture of past or ongoing exposure over extended time.
c. At present, blood lead is the most useful and practical biological exposure monitor although shortcomings may exist in what it tells about lead behavior in inaccessible tissue sites.

As an example of a problem with blood lead, Piomelli et al. (1984) show that if one tests circulating body lead in children by the technique of provocative chelation, i.e., a single treatment with a lead binding agent that removes lead and produces lead excretion in urine, this urinary lead amount in a percentage of children with moderately elevated Pb-B levels (25 to 30 μg/dl) is equivalent to levels in those individuals with higher Pb-B levels and who require hospitalization and treatment. In other words, if only Pb-B levels were relied on, the actual toxicity risk due to toxic, circulating lead might be underestimated.

IV. ADVERSE HEALTH EFFECTS OF LEAD: RELATIONSHIP TO PUBLIC HEALTH RISK AND SOCIETAL WELL-BEING

Section 118(f) of SARA directs that there be an evaluation of the long-term adverse consequences of ongoing lead exposure at levels currently being encountered by children in the United States. Both the individual and society as a whole are damaged by adverse health effects associated with lead exposure. Lead-induced reductions in IQ, for instance, not only place the individual at a disadvantage, but also eventually place the nation at a collective disadvantage in an increasingly competitive, technical, and cognition-intensive world economy. While the aggregate social cost of lead poisoning in dollar terms is not considered in this report, the reader can refer to a detailed assessment of such costs recently carried out by U.S. EPA (1985).

This chapter is organized into four sections. Section A explains why the concentration of lead in blood (Pb-B) is typically used as the measure of exposure in assessing the health effects of lead. Section B describes the various adverse health effects of lead in children that occur at different levels of lead exposure, with special emphasis on effects that have been found at prevalent levels of population exposure.

Section C summarizes current information relating the primary adverse health effects of lead to the lowest exposure levels (in terms of Pb-B concentrations) at which the effects have been reliably observed to occur. Given the progressive decline in the "lowest-observed-effect-levels" throughout the history of lead research, continued work may reveal even lower levels for lead-induced toxicity in the future. Section C also discusses adverse health effects in terms of population risk. Section D discusses the relative persistence of various effects of lead and considers criteria for defining "persistence of effect."

A. THE EXPOSURE (DOSE) INDEX IN ASSESSMENT OF THE ADVERSE HEALTH EFFECTS OF LEAD IN HUMAN POPULATIONS

The concentration of lead in whole blood (Pb-B) has been the most commonly used indicator of exposure in studies relating lead exposure to toxicological and health endpoints. Blood is readily accessible and relatively convenient to obtain. Most other organs and tissues of the body are not as easily sampled. Lead can be accurately, precisely, and reliably measured at quite low concentrations in blood, thus providing an important quantitative index that can be related to both external exposure levels and the toxicological or health effects induced by lead. As a link to external exposure, Pb-B levels offer a more direct index of the actual amount of lead that has entered the body. In obtaining quantitative dose-response relationships between lead and biological effects, Pb-B levels have been extremely useful. Indeed, the robustness of these relationships has resulted in the clinical use of Pb-B levels as an indicator of lead-induced injury to organs and tissues (Chisolm and Barltrop, 1979).

There are also limitations in Pb-B levels that should be recognized. The relationship between external and internal exposure measures is not linear over the entire range of possible values. For example, Pb-B levels are proportionately not as elevated at relatively high environmental levels as one would expect from their values at lower environmental levels. The Pb-B levels associated with specific types of biological effects also vary for individuals and populations. Children generally show a much greater vulnerability to toxic effects at a given Pb-B level than adults do at the same level.

As an indicator of the total body burden of lead, Pb-B levels may underrepresent the actual body burden. In a detailed study of children who underwent provocative chelation testing, i.e., biochemical removal of lead from the body, Piomelli et al. (1984) found that a sizable number of children who had relatively low Pb-B levels nevertheless had high levels of lead in other compartments of the body. Since lead accumulates over time, indicators of cumulative exposure, such as the amount of lead in teeth or bones, offer certain advantages over Pb-B levels. On the other hand, exposure is not static in children, and an indicator that responds relatively rapidly to abrupt or intermittent changes in their intake of lead (e.g., through ingestion of lead paint chips) is valuable.

On the whole, Pb-B level remains the scientifically and diagnostically most useful index of internal body exposure to lead. The following sections therefore rely heavily on this important variable in presenting the current understanding of adverse health effects in children.

B. MAJOR ADVERSE HEALTH EFFECTS OF LEAD IN CHILDREN

There is a wealth of information about the variety and the relative intensity of adverse effects of lead on different tissues and organ systems of children, including those considered as the critical target organs of lead toxicity. Such effects involve many levels of physiological and anatomical organization within the body, starting with the cellular/subcellular level and progressing to higher levels of functional and structural organization. At a sufficient level of lead exposure, virtually all body systems will be injured or have a high risk of injury. The full spectrum of such effects is presented in a comprehensive assessment of this topic in Air Quality Criteria for Lead (U.S. EPA, 1986a). In this chapter the primary focus is on those effects that occur at relatively low or prevalent chronic levels of lead exposure in the United States.

1. Effects of Lead on Heme Biosynthesis, Erythrocyte Physiology and Function, and Erythropoietic Pyrimidine Metabolism

Effects on the blood's biochemical functions are interrelated and have variable biological impact. Heme, the iron-containing prosthetic or cofactor group, is critical to the basic function of various heme proteins in cells in many different organ systems. These organs include the blood-forming tissue and the liver, kidney, and brain. In addition to the direct effects of lead on heme biosynthesis, there are potentially significant indirect impacts on the central nervous system, caused by the lead-induced accumulation of delta-aminolevulinate (ALA), a potential neurotoxicant (discussed in U.S. EPA, 1986a).

Figure IV-1 depicts graphically the various steps in the heme biosynthetic pathway that are known to be affected by lead, although the actual mechanisms may not be fully understood in all cases. These steps include the feedback derepression (stimulation) of ALA-synthetase activity, the inhibition of

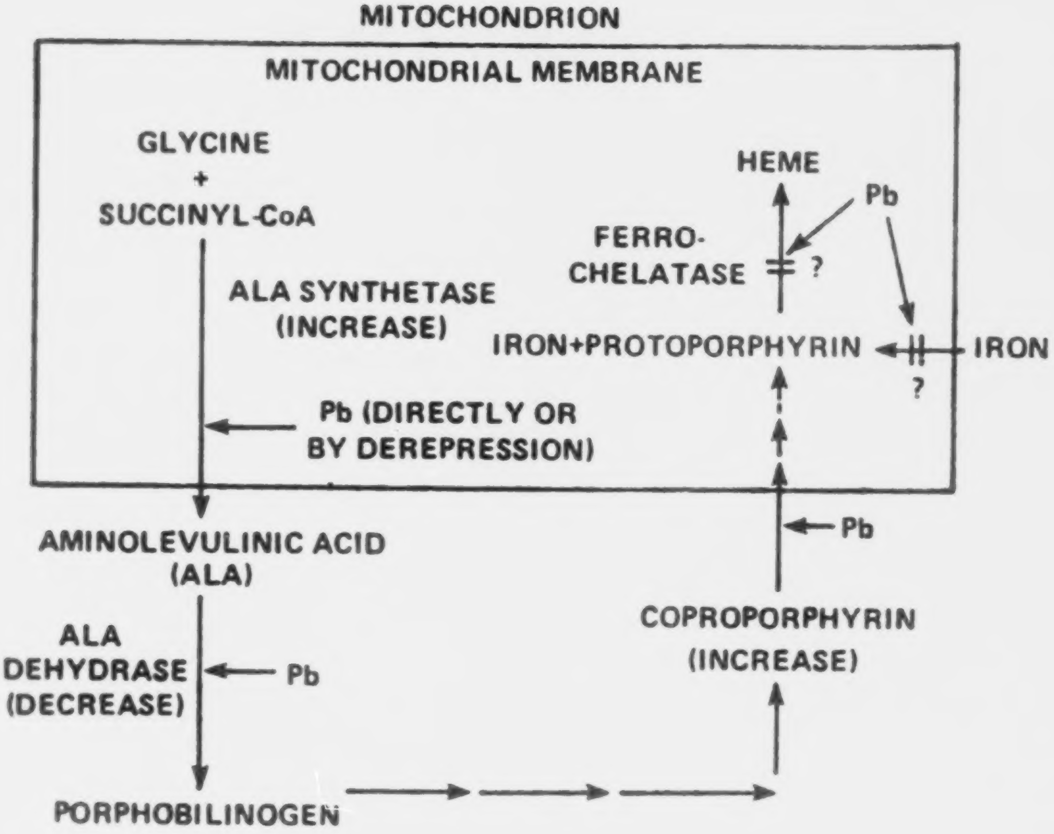

MITOCHONDRION
MITOCHONDRIAL MEMBRANE
GLYCINE
+
SUCCINYL-CoA
ALA SYNTHETASE
(INCREASE)
Pb (DIRECTLY OR
BY DEREPRESSION)
HEME
Pb
FERRO-
CHELATASE
?
IRON+PROTOPORPHYRIN
IRON
?
Pb
AMINOLEVULINIC ACID
(ALA)
ALA
DEHYDRASE
(DECREASE)
Pb
COPROPORPHYRIN
(INCREASE)
PORPHOBILINOGEN

Figure IV-1. Effects of lead (Pb) on heme biosynthesis.

Source: EPA (1986a).

ALA-dehydratase (porphobilinogen synthetase) activity, coproporphyrin utilization, and the insertion of iron into protoporphyrin IX to form the prosthetic group, heme. Companion effects on the uroporphyrins, which are mainly affected at relatively high exposure levels in humans, are also presented.

The accumulation of protoporphyrin IX (zinc protoporphyrin, ZPP; protoporphyrin in erythrocytes, EP) is not only an indicator of diminished heme biosynthesis, but also signals general mitochondrial injury, since the final step in heme biosynthesis, which includes EP, occurs in the mitochondria. Such subcellular injury may impair a variety of processes, including cellular energetics and calcium homeostasis. The various effects of lead within the heme biosynthesis pathway are shown in Table IV-1. Note that these are qualitative in nature. Quantitative relationships are presented in Section C of this chapter.

TABLE IV-1. EFFECTS OF LEAD ON THE HEME BIOSYNTHESIS PATHWAY IN HUMANS

Step Affected	Results	Comments
Inhibition of ALA-D activity	Accumulation of ALA in the body and excretion.	Plasma and urine levels rise; ALA itself may be neurotoxic at higher levels.
Feedback derepression of activity of ALA-synthetase	Accumulation of ALA in the body and excretion.	Much less sensitive than ALA-D activity; occurs at Pb-B >40 μg/dl.
Inhibited conversion of coproporphyrin	Accumulation of coproporphyrin in urine.	Inhibition of coproporphyrin utilization; indicates ongoing lead exposure.
Inhibited conversion of protoporphyrin IX (EP) to heme	Accumulation of EP in red blood cells.	Sensitive measure of lead toxicity; elevation indicates mitochondrial injury.

Figure IV-2 graphically depicts lead-associated disturbances of the body heme pool. Most effects are documented, while some are only suggested by available experimental data. All of these effects can be summarized as follows:

(1) Hemoglobin effects--Lead can disturb the biosynthesis of hemoglobin, the general oxygen transport substance in mammals, leading, in a dose-dependent manner, to anemia and potential exacerbation of hypoxic responses to other toxic agents.

(2) Neural effects--Lead can reduce the amount of nervous system hemoproteins available for brain cellular energetics and development in neurons, axons, and glia.

(3) Renal and endocrine effects--Lead can disturb heme-mediated generation of the important hormonal metabolite of vitamin D, 1,25-$(OH)_2$-vitamin D. This hormone serves a number of metabolic functions in humans, including regulation of calcium metabolism and function.

(4) Hepatic effects--Lead can impair the ability of heme-dependent liver enzyme systems to adequately detoxify foreign substances.

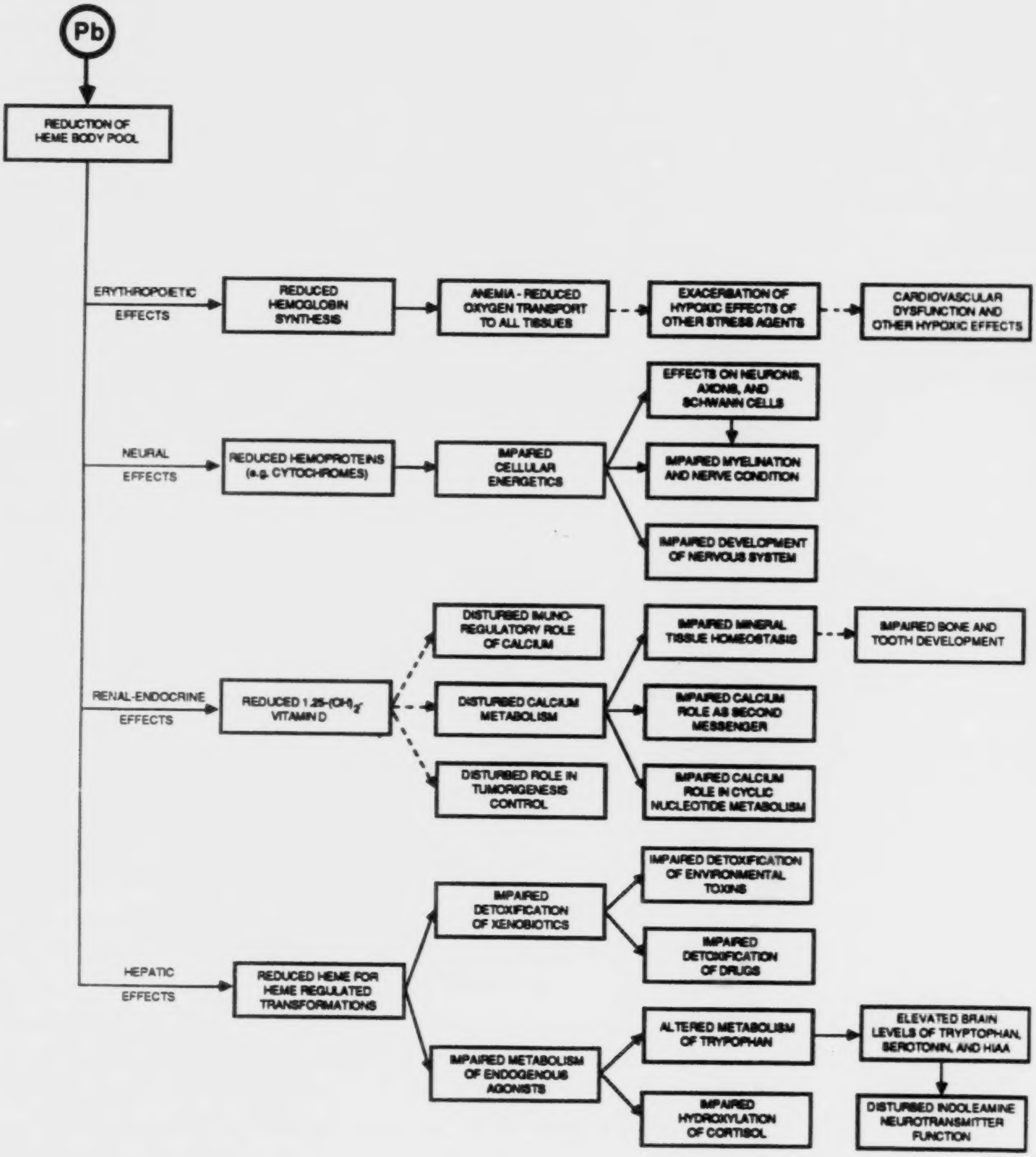

Pb
REDUCTION OF HEME BODY POOL
ERYTHROPOIETIC EFFECTS
REDUCED HEMOGLOBIN SYNTHESIS
ANEMIA - REDUCED OXYGEN TRANSPORT TO ALL TISSUES
EXACERBATION OF HYPOXIC EFFECTS OF OTHER STRESS AGENTS
CARDIOVASCULAR DYSFUNCTION AND OTHER HYPOXIC EFFECTS
NEURAL EFFECTS
REDUCED HEMOPROTEINS (e.g. CYTOCHROMES)
IMPAIRED CELLULAR ENERGETICS
EFFECTS ON NEURONS, AXONS, AND SCHWANN CELLS
IMPAIRED MYELINATION AND NERVE CONDITION
IMPAIRED DEVELOPMENT OF NERVOUS SYSTEM
RENAL-ENDOCRINE EFFECTS
REDUCED 1,25-(OH)2- VITAMIN D
DISTURBED IMUNO-REGULATORY ROLE OF CALCIUM
DISTURBED CALCIUM METABOLISM
DISTURBED ROLE IN TUMORIGENESIS CONTROL
IMPAIRED MINERAL TISSUE HOMEOSTASIS
IMPAIRED BONE AND TOOTH DEVELOPMENT
IMPAIRED CALCIUM ROLE AS SECOND MESSENGER
IMPAIRED CALCIUM ROLE IN CYCLIC NUCLEOTIDE METABOLISM
HEPATIC EFFECTS
REDUCED HEME FOR HEME REGULATED TRANSFORMATIONS
IMPAIRED DETOXIFICATION OF XENOBIOTICS
IMPAIRED DETOXIFICATION OF ENVIRONMENTAL TOXINS
IMPAIRED DETOXIFICATION OF DRUGS
IMPAIRED METABOLISM OF ENDOGENOUS AGONISTS
ALTERED METABOLISM OF TRYPOPHAN
ELEVATED BRAIN LEVELS OF TRYPTOPHAN, SEROTONIN, AND HIAA
DISTURBED INDOLEAMINE NEUROTRANSMITTER FUNCTION
IMPAIRED HYDROXYLATION OF CORTISOL

Figure IV-2. Multi-organ impact of reductions on heme body pool by lead. Impairment of heme synthesis by lead results in disruption of a wide variety of important physiological processes in many organs and tissues.

Source: EPA (1986a).

In addition to its effects on heme biosynthesis, lead has related effects on the cellular health and function of the red blood cell, inducing deleterious changes, such as enhanced fragility and higher rates of lysis. Such cell destruction can result in enough hemolytic loss of hemoglobin to significantly augment the lead-induced anemia that occurs through other routes. Lead-induced disturbances in red blood cell formation and maturation also occur by way of alterations in pyrimidine metabolism. Inhibition of the enzyme pyrimidine-5′-nucleotidase (Py-5′-N) impairs the degradation of large nucleic acid biomolecules and interferes with normal cellular energetics involved in the formation of the mature erythrocyte.

2. Neurotoxic Effects of Lead in Children

Although lead has diverse health effects, its neurotoxic effects in children are particularly notable because of the sensitivity of the developing nervous system. Indeed, the central nervous system is the primary target organ for lead toxicity in children. In the earlier part of this century, childhood lead neurotoxicity was primarily recognized as the result of acute, high-level lead exposure resulting, for example, from the ingestion of leaded paint chips. Prior to the introduction of chelation therapy, which biochemically removes a portion of lead from the body, severe lead poisoning with encephalopathy had a mortality rate of 65% (NAS, 1972). Since the advent of chelation treatment, mortality rates have declined significantly. In the United States, the use and refinement of chelation therapy in lead poisoning owes much to the work of Chisolm and colleagues in Baltimore (NAS, 1972).

The Pb-B levels associated with such severe poisoning were quite high, but were also quite variable, reflecting individual differences in vulnerability and varying times between the exposure episode and clinical intervention. Children surviving acute poisoning episodes, with or without manifest encephalopathy, were often found to have severe neurological sequelae, traced to permanent damage to the central nervous system. Observations by Byers and Lord (1943) and other clinicians showed that children manifested mental retardation, seizures, optic atrophy, sensory-motor deficits, and behavioral dysfunctions long after their acute poisoning experiences. Perlstein and Attala (1966) reported such sequelae in 37% of children who suffered lead poisoning without evidence of encephalopathy.

The history of research on lead has shown a progressive decline in the lowest exposure levels at which neurotoxic and other effects can be detected. Consequently, the attention of the medical and research community has largely shifted to the effects of chronic, low-level lead exposure (although cases of severe poisoning still occur). Epidemiologic studies have been the primary means of identifying the effects of prevalent lead exposure levels on populations of children. These studies have been of two general types: (1) cross-sectional or retrospective studies, which examine variables at a particular time point in the present or reconstructed past, and (2) longitudinal or prospective studies, which measure variables over an extended period of time into the future.

Longitudinal or prospective designs have a number of advantages for the study of environmental pollutants. In the case of lead, a key advantage of prospective studies is that one can evaluate each subject's pattern of lead exposure with more accuracy and precision than one can with cross-sectional or retrospective studies. Information on the history of exposure is obviously important in assessing the effects of a cumulative toxicant on endpoints such as neurobehavioral functions, which may reflect alterations induced during critical periods of earlier neurological development.

In the subsections that follow, important recent findings from a group of prospective studies are discussed first, followed by a summary of findings from cross-sectional and other investigations of childhood lead neurotoxicity.

a. Prospective Studies of Lead Neurotoxicity

Findings from a group of well-conducted studies now indicate that disturbances in early neurobehavioral development occur at levels well below those considered "safe" or even "normal" in recent years. The most clearly identified effect thus far has been lower scores on the Mental Development Index of the Bayley Scales of Infant Development, a well-standardized test of infant intelligence. Other developmental endpoints, such as shorter gestational age and lower birth weight, have also been associated with prenatal lead exposure in many of these studies, but are discussed separately below in Section 3 ("Other Adverse Health Effects"). A recent interpretive review of these studies (Davis and Svendsgaard, 1987) has assessed in greater detail the features of the studies and their implications for public health.

A number of prospective studies are currently underway in various parts of the world, but those conducted in Boston, Cincinnati, and Cleveland, in the United States, and Port Pirie, South Australia, have progressed far enough to provide published results that can be critically assessed and interpreted. As noted by Davis and Svendsgaard (1987), these four studies collectively provide evidence that is much stronger and more compelling than any single study or past studies in general could provide.

Among the strengths of these studies are the fact that, although independently conducted, they have benefited from the exchange of information and ideas among the principal investigators concerning the design and analysis of their work (Bornschein and Rabinowitz, 1985). Also, as a group, these studies are much more sophisticated methodologically than most previous work. For example, they consider and employ appropriate controls for many more covariates and possible confounders, and with study populations numbering in the hundreds, they have greater statistical power to detect subtle effects than most earlier studies had. Their analytic methods for measuring blood lead levels are quite accurate and reliable. Moreover, by consistently using the Bayley Scales and certain other outcome measures, they have assessed developmental effects in a manner that permits a direct comparison of their results. Thus, more confidence may be placed in the conclusions and weight of evidence provided by this body of work.

Table IV-2 summarizes some of the key features of the four prospective studies reviewed below. It may be useful to note that the Bayley Scales of Infant Development comprise three indexes of mental, motor, and emotional development. Of particular importance here is the Mental Development Index (MDI), which was designed to assess: "sensory-perceptual acuities, discriminations, and the ability to respond to these; the early acquisition of 'object constancy' and memory, learning, and problem-solving ability; vocalizations and the beginnings of verbal communication; and early evidence of the ability to form generalizations and classifications, which is the basis of abstract thinking" (Bayley, 1969, p.3). The scales have a mean of 100 and a standard deviation of 16.

The first prospective study to report effects of prenatal lead exposure on later postnatal development was conducted in Boston by Bellinger et al. (1984). Blood lead levels were measured for 249 umbilical cords of infants born to

TABLE IV-2. SUMMARY OF MAJOR FINDINGS FROM PROSPECTIVE STUDIES OF EARLY DEVELOPMENTAL EXPOSURE TO LEAD[a]

Population Average Pb-B (μg/dl)	Time of Pb-B Measurement	Endpoint (Amount of Deficit)[b]	Population	Reference
14.0	Postnatal (6-mo.)	MDI[c] 24 mo. (2 pts)	Port Pirie, S. Australia	Vimpani et al. (1985, 1987)
6.5	Birth (cord)	MDI at 6, 12, 18, 24 mo. (4-8 pts)	Boston, MA	Bellinger et al. (1984, 1985, 1986, 1987a)
6.8	Postnatal (24 mo.)	McCarthy Scales at 57 mo. (3 pts)	Boston (continued)	Bellinger et al. (1987b)
8.0	Prenatal (maternal)	MDI at 6, 12 mo. (8 pts)	Cincinnati, OH	Dietrich et al. (1986, 1987a,b)
5.8	Birth (cord)	MDI at 12 mo. via Neurological Soft Signs at birth	Cleveland, OH	Wolf et al. (1985) Ernhart et al. (1985a, 1986)

[a] Source: U.S. EPA (1986a), with updating.

[b] Deficit per 10-μg/dl (approximate) increment in Pb-B, except for difference in MDI scores for high- and low-lead groups in first stages of Boston Study; amount of deficit not stated for Cleveland study.

[c] MDI = Bayley Mental Development Index

middle- to upper-middle class parents. The use of higher socioeconomic-status (SES) subjects complements the focus on lower SES subjects in most other lead studies. Multivariate regression analyses for 216 subjects showed an association between cord blood lead levels and performance on the Bayley MDI at six months of age. The covariate-adjusted difference between low ($\bar{X}$ = 1.8 µg/dl) and high ($\bar{X}$ = 14.6 µg/dl) lead exposure groups was nearly 6 points on the MDI. Continued follow-up of these subjects has shown that a 4-8 point deficit in MDI scores persisted at 12, 18, and 24 months of age (Bellinger et al., 1985, 1986, 1987a). No effect was evident using postnatal blood lead levels.

More recently, Bellinger et al. (1987b) have reported that the Boston cohort of children also show cognitive deficits on the McCarthy Scales of Children's Abilities at about 5 years of age. Although initial analyses do not indicate that these deficits can be attributed to prenatal lead exposure (after adjusting for the influence of potentially confounding factors), they do show a significant relationship to earlier blood lead levels (at 24 months) rather than concurrent levels.

Dietrich et al. (1986, 1987a,b) enrolled 305 pregnant women from the inner city of Cincinnati and measured their blood lead levels at the first prenatal clinic visit ($\bar{X}$ = 8.0 µg/dl for 245 subjects). Blood lead concentrations of the newborn infants were determined at postnatal day 10 ($\bar{X}$ = 4.5 µg/dl for 280 subjects). Regression analyses (including a statistical technique known as structural equation modeling) indicated that prenatal lead exposure was inversely related to male infants' scores on the 6-month Bayley MDI as well as the Psychomotor Development Index (PDI).

These effects were both direct and indirect; that is, prenatal lead levels were not only directly related to impaired performance on the Bayley Scales, but were also related to reduced gestational age and reduced birth weight, which in turn were associated with lower MDI and PDI scores. The total direct and indirect effects of prenatal lead exposure on MDI scores amounted to approximately 8 points deficit for every 10-µg/dl increase in blood lead level (Dietrich et al., 1987b). A similar association was found between MDI scores and neonatal blood lead, but preliminary analyses using postnatal blood lead measurements at 3 and 6 months indicated no significant relationships (Dietrich et al., 1986b). Thus, as in the Boston study, prenatal rather than postnatal lead exposure had the predominant influence on postnatal neurobehavioral performance. Interestingly, however, these effects were confined to the males in the

Cincinnati study. It is important to note that the statistical significance and the magnitude of the effects described here were already adjusted for various factors such as maternal alcohol and tobacco usage, home environment, and SES. More recent analyses of 12-month data for this same cohort have indicated that deficits in the MDI and other effects persist at least through the first year of life (Dietrich et al., 1987c), which, again, is consistent with findings from the Boston study.

Reports from Ernhart et al. (1985a, 1986, 1987a) have also addressed the issue of prenatal lead exposure and postnatal neurobehavioral function as examined in a prospective study in Cleveland, Ohio. Maternal and cord blood samples were obtained at the time of delivery from 185 mothers ($\bar{X}$ = 6.5 µg/dl) and 162 infants ($\bar{X}$ = 5.8 µg/dl). Of these, only 132 samples were actual mother-infant pairs. The infants were evaluated on the Brazelton and the Graham-Rosenblith neonatal behavioral assessment scales, which showed three significant effects (out of 17 outcomes examined) related to blood lead: abnormal reflexes, neurological soft signs, and muscle tonus. Using the restricted data set of 132 mother-infant pairs, only neurological soft signs were significantly related to cord blood lead. A brief report (Wolf et al., 1985) on later outcomes in this same cohort mentioned a statistically significant relationship between the neurological soft signs measure and the Bayley MDI scores at 12 months. Thus, despite the comparatively small number of subjects in this study, it appears, as noted by Davis and Svendsgaard (1987), that neurobehavioral effects of quite low prenatal lead exposure were detected at birth and that, indirectly, 12-month Bayley MDI scores may have declined as a result.

Another major prospective study is under way in Port Pirie, South Australia. Preliminary results of testing 592 children on the Bayley MDI and PDI scales at 24 months of age have been reported by Vimpani et al. (1985) and Baghurst et al. (1987). Multiple regression analyses indicated that lead exposure was significantly related to reduced MDI scores. For every 10-µg/dl increase in blood lead, scores on the 24-month MDI dropped an average of about 2 points, which is notably consistent with findings from the Boston and Cincinnati studies. However, unlike the latter studies, the strongest relationship in Port Pirie was found using postnatal blood lead.

Blood lead levels rose sharply in the Port Pirie cohort from about 14 µg/dl at 6 months of age to approximately 21 µg/dl at 15 months. Davis and Svendsgaard (1987) suggest that earlier testing on the Bayley Scales (e.g., at 6 months) might have revealed a stronger effect of prenatal exposure than could be detected later, after blood lead levels had increased so much between 6 and 15 months. More recent, but still preliminary, analyses incorporating controls for maternal intelligence and quality of the home environment indicated that the blood lead-MDI relationship was "markedly attenuated" when home environment measures were included in the multivariate regressions (Vimpani et al., 1987). Nevertheless, the association between 6-month blood lead levels and 24-month MDI scores remained statistically significant.

Other prospective studies are being conducted by McBride et al. (1987) in Sydney, Australia, by Rothenberg et al. (1987) in Mexico City, Mexico, by Graziano et al. (1987) in Titova Mitrovica, Yugoslavia, and by Moore et al. (1987) in Glasgow, Scotland. The results from these studies are still preliminary or have not yet been reported in sufficient detail to allow critical evaluation.

In sum, the studies for which adequate information is available are remarkably consistent in identifying a link between low-level lead exposure during early development and later neurobehavioral performance as reflected in deficits on the Bayley Mental Development Index. Moreover, the studies generally point to the prenatal period of exposure as the most critical, although postnatal exposure may still be important and even override the effect of prenatal exposure under some conditions. Blood lead levels of 10 to 15 μg/dl, and possibly lower, constitute a level of concern for these effects (Davis and Svendsgaard, 1987; U.S. EPA, 1986a). The public health implications of a 2- to 8-point decline in Bayley MDI scores have been examined by Davis and Svendsgaard (1987) and Grant and Davis (1987). They noted that, although a change of a few points in the MDI for an individual child may not be clinically significant, a 4-point downward shift in a normal distribution of MDI scores for a population of children would result in 50% more children scoring below 80.

b. Cross-Sectional and Other Studies of Lead Neurotoxicity

A great deal of important and useful information on the neurotoxic effects of lead in children has been provided by other epidemiological studies. A detailed and comprehensive evaluation of this body of work may be found in U.S. EPA (1986a). Starting in the early 1970s, several studies were devoted to assessing the relationship between variables such as IQ and blood lead levels in various populations of children with known exposure to lead (e.g., residents of smelter communities or inner-city children identified through lead screening programs). By today's frame of reference, exposure levels for these subjects were quite high, with mean levels well above 50 µg/dl in many instances. For example, de la Burde and Choate (1972) found various neurobehavioral dysfunctions and IQ deficits in children whose Pb-B levels averaged 58 µg/dl at the time of assessment. Later follow-up (de la Burde and Choate, 1975) indicated that the dysfunctions and deficits persisted. A variety of similar neurobehavioral impairments were also evident in both post-encephalopathic children (Rummo, 1974; Rummo et al., 1979) and "asymptomatic" children (Kotok et al., 1977).

The difficulties in drawing conclusions about the likelihood of causal relationships from these early studies are illustrated by the work of Perino and Ernhart (1974), who found an association between lower IQ scores and Pb-B levels in children identified through the New York City lead screening program. Follow-up investigation of these children, with control for parental education, led Ernhart et al. (1981) to conclude that the effect they had first reported was probably not due to lead or represented at best only a minimal effect on intelligence. Furthermore, after reanalysis of their earlier data, Ernhart et al. (1985b) concluded that their results provided no indication of an effect of lead on intelligence in the children they had examined.

Despite the limitations of the early epidemiologic investigations, their findings pointed the way for later studies at lower exposure levels. An important pioneering study of a general population of children without known elevated lead exposure was conducted by Needleman et al. (1979). The deciduous teeth of subjects were analyzed for lead content, which served as an indicator of cumulative lead exposure for the more than 2,000 children enrolled in the study. Based on classroom teacher ratings of the behavior of these children, an apparent dose-response relationship was found. More detailed analyses, taking into account various confounding variables, showed significant differences in IQ and certain other neurobehavioral measures for 58 high-lead children versus 100 low-lead children. Later analyses of the data from this study extended the findings and their implications. For example, Needleman et al. (1982) noted that a difference of 4 points in mean verbal IQs for the high- and low-lead groups meant that children in the high-lead group were three times as likely to have a verbal IQ below 80, and no high-lead children scored in the superior IQ range (>125). Follow-up investigation of the same children's school performance indicated that, four years later, high-lead children were significantly more likely to have been retained in grade (Bellinger et al., 1984b).

Smith et al. (1983) investigated the relationship between lead levels in teeth and measures of behavior and intelligence in over 400 British children, but found that, after correcting for social class, home quality, and other confounding factors, the association between lead and IQ or academic performance was not statistically significant. Tooth-lead levels in these children were significantly below those reported in other industrialized countries.

Similar findings were reported by Harvey et al. (1984) for almost 200 British children with low Pb-B levels (mean: 15.5 µg/dl): after adjusting for confounding variables such as social class, the association between blood lead and IQ was no longer significant. Social class may have also confounded the positive results of Yule and his colleagues in their studies of British children. In their first study, IQ was reduced as a function of increasing blood lead level (Yule et al., 1981), but a better controlled replication study (Lansdown et al., 1986) showed no significant effect of lead on IQ. Other work by Yule and Lansdown (1983) and Hunter et al. (1985) showed no significant effect on IQ but did show significant effects on reaction time, consistent with findings by Needleman et al. (1979). Similarly, teacher ratings of high-lead children (Pb-B levels: 12 to 26 µg/dl) indicated behavioral impairments in line with the earlier findings of Needleman et al. (1979).

A series of studies in Germany by Winneke et al. (1982, 1983, 1984) parallel the British findings in several respects. Social variables appeared to play an important role in the associations between neurobehavioral function and lead exposure. With mean blood lead levels below 15 µg/dl, IQ scores were not significantly reduced, but reaction time performance and certain other neurobehavioral functions did show significant impairments (Winneke et al., 1984).

Although the British and German studies show few if any significant associations between low-level lead exposure and cognitive function after controlling for social class, the findings are consistent in the direction of their effect and compatible with an overall dose-response relationship, with these studies falling at the low end of the lead-IQ relationship (U.S. EPA, 1986a). It may be, as Pocock et al. (1987) have concluded, that lead has little or no effect on IQ as measured in the British studies. But it is also quite possible that IQ tests or other aspects of the design and analysis of these studies are inadequate to detect lead-induced neurological impairments at the relatively low exposure levels involved (Smith, 1985).

Other recent studies provide more suggestive evidence that lead exposure at such relatively low levels may in fact be significantly associated with deficits in IQ. For example, Schroeder and Hawk (1987), in replicating earlier work (Schroeder et al., 1985), found a highly significant linear relationship between IQ and blood lead level over a range of 6 to 47 µg/dl in a group of 75 black children. Since these children were all of low socioeconomic status, SES was not a confounder in this study. Studies by Fulton et al. (1987) in Edinburgh, Scotland, and by Hatzakis et al. (1987) in Lavrion, Greece also provide strong evidence of IQ deficits related to children's lead exposure at blood lead levels below 25 µg/dl.

Considered singly, none of the above studies can provide definitive evidence that low-level lead exposure is linked to reduced cognitive performance in children. However, Needleman (1987) recently reported the results of a meta-analysis of 13 such studies and noted that the joint probability of obtaining the reported results was less than 3 in a billion. Thus, the overall pattern of findings supports the conclusion that low-level lead exposure is related to neurobehavioral dysfunction in children.

In addition to the above assessments of the relationship of lead to cognition and behavior, other aspects of lead-associated neurotoxicity have been examined. Burchfiel et al. (1980) examined components of the EEG profiles for a subset of children studied by Needleman et al. (1979) and found significant differences in EEG activity in the higher dentine-lead group.

Otto and his co-workers (Otto et al., 1981; Benignus et al., 1981; Otto et al., 1982, 1985) have also evaluated neurophysiological function in relation to blood lead levels in children. Using various test paradigms, they have found disturbances in features of the EEG that correlate with Pb-B levels. In some cases, these effects appeared to persist for 2 to 5 years. These investigators have also reported electrophysiological alterations measured through auditory brainstem evoked potentials (e.g., Robinson et al., 1985, 1987). In addition, evidence of lead-related reduced hearing acuity has been reported by Robinson et al. (1985), supported by an analysis of audiometric data from NHANES II for children aged 4 to 19 years (Schwartz and Otto, 1987). The relationship between Pb-B level and hearing threshold was highly significant ($p < 0.0001$) for the large dataset from NHANES II. As noted by the authors, such impairment of hearing could contribute to other reports of neurobehavioral deficits such as learning disabilities and poor classroom behavior.

3. Other Adverse Effects of Lead on the Health of Young Children

Even a cursory review of the myriad effects of lead on children is beyond the scope of this report. For a more comprehensive evaluation, the reader is referred to the Air Quality Criteria Document for Lead (U.S. EPA, 1986a). However, certain recent findings related to child growth and development are summarized here because of their statistical and public health significance.

In addition to neurobehavioral endpoints, the prospective studies described above have examined various outcomes related to fetal and postnatal growth and maturation. In the Port Pirie study, McMichael et al. (1986) enrolled 831 pregnant women and followed 774 of the pregnancies to completion. Multivariate analysis showed that pre-term deliveries (before the 37th week of pregnancy) were significantly related to maternal blood lead at delivery. If late fetal deaths were excluded, the association was even stronger: the relative risk of pre-term delivery at exposure levels of 14 µg/dl or greater was 8.7 times the risk at levels up to 8 µg/dl.

The Cincinnati prospective study has also noted an effect of prenatal lead exposure on the duration of gestation. As mentioned above, Dietrich et al. (1986, 1987a,b) found that declines in Bayley scores were mediated in part by reduced gestational age associated with lead exposure. The effect amounted to about a half-week's reduction in gestation for about every 10-µg/dl increment in blood lead. Note that this effect was detected despite the fact that infants of less than 35 weeks gestational age were excluded from the Cincinnati study. Similarly, the Boston study (Bellinger et al., 1984) excluded infants of less than 34 weeks gestational age. As noted by Davis and Svendsgaard (1987) in their review of these findings, such criteria would make it more difficult to detect an effect on duration of gestation.

Gestational age was also shown to be significantly reduced as a function of increasing cord or maternal blood lead levels in a well-conducted cross-sectional study of 236 mothers and their infants in Glasgow, Scotland (Moore et al., 1982). The geometric mean blood lead level for the mothers was approximately 14 µg/dl and for the infants was approximately 12 µg/dl.

The clearest evidence concerning an effect of lead on birth weight and growth comes from the Cincinnati study. Preliminary analyses by Bornschein et al. (1987a,c) have indicated that, for approximately every 10-µg/dl increment in blood lead, the decrease in birth weight ranged from 58 to 601 grams, depending on the age of the mother. Birth length also appeared to be significantly related to maternal blood lead (-2.5 cm per log unit blood lead), although the effect was evident only in white infants.

Other prospective studies are suggestive but less definitive regarding birth weight and fetal growth. Bellinger et al. (1984) reported data showing an exposure-related trend in the percentage of small-for-gestational age infants in the Boston study, but the differences fell just short of statistical significance at $p = 0.05$. In Port Pirie, the proportion of low-birth-weight deliveries was more than double that outside Port Pirie (respective maternal blood lead levels: 10.4 vs. 5.5 µg/dl). Yet within both groups, low-birth-weight pregnancies were (nonsignificantly) associated with lower blood lead levels. Also, regression analyses indicated no evidence of intrauterine growth retardation using "small-for-dates" data. To complicate matters further, head circumference was significantly inversely related to maternal blood lead, but crown-heel length showed no association with lead exposure.

Other findings from the Port Pirie study are pertinent here. Of the 23 miscarriages in this study, all but one occurred in the more highly exposed Port Pirie mothers. Also, 10 of 11 stillbirths occurred to Port Pirie women. Specifically, the proportion of stillbirths was 17.5/1000 live births in Port Pirie versus 5.8/1000 outside Port Pirie and 8.0/1000 for all of South Australia. Nevertheless, the average maternal blood lead level at delivery was significantly lower for stillbirths (7.9 µg/dl) than for live births (10.4 µg/dl). As noted by Davis and Svendsgaard (1987), these anomalous findings for birth weight and stillbirths in the Port Pirie study suggest the possibility that the fetus and/or placenta was acting as a "sink" for the mother's body burden in such cases.

Congenital malformations were also considered in the Port Pirie study, but no significant relationship to lead exposure was found. Unfortunately, too little information was presented on this aspect of the Port Pirie study by McMichael et al. (1986) to judge the strength of their conclusions. Although Ernhart et al. (1986, 1987b) also reported no significant relationship between lead exposure and congenital malformations for the Cleveland study, that study had a comparatively small number of subjects and a limited range of blood lead values, which would have made it difficult to detect an effect of lead if one existed. However, a retrospective study by Needleman et al. (1984) did report an association between cord blood lead and the occurrence of minor malformations in 4,354 infants born in Boston. The effect was significant only for minor malformations (e.g., hemangiomas/lymphangiomas, hydroceles, skin tags, papillae, undescended testicles) taken as a whole, not for any single malformation. Also, unexpected significant reductions in first trimester bleeding, premature labor, and neonatal respiratory distress were found to be associated with higher prenatal lead exposure.

Although no definitive judgment can be reached at this point regarding the possible teratogenic effects of low-level lead exposure as far as congenital malformations are concerned, other effects of lead on fetal development seem more clearcut. As concluded by Davis and Svendsgaard (1987), the weight of available evidence suggests that the duration of gestation is affected by exposure to lead during pregnancy and that such effects can occur at blood lead levels below 15 µg/dl. In addition, birth weight and possibly other aspects of fetal growth appear to be reduced by prenatal lead exposure levels of less than 15 µg/dl. Recent analyses also suggest that delays in developmental milestones (e.g., age of first sitting up, walking, or speaking) are related to Pb-B levels in children (Schwartz and Otto, 1987).

Later growth also appears to be affected by lead exposure postnatally. Schwartz et al. (1986) have recently reported that an analysis of the NHANES II dataset revealed significant relationships between Pb-B levels and height ($p < 0.0001$), weight ($p < 0.001$), and chest circumference ($p < 0.026$) in young children (<7 years old). These growth milestones were inversely related to Pb-B levels over the range of 5 to 35 µg/dl. Work in Belgium by Lauwers et al. (1986) also points to a relationship between children's lead exposure and disturbances in physical growth up to about 8 years of age. Although a number of potentially confounding variables were considered in these studies, a more definitive epidemiologic design would be a prospective study. Preliminary analyses of data for 260 infants from the Cincinnati prospective lead study (Shukla et al., 1987) in fact indicate that covariate-adjusted growth rates are significantly related to postnatal increases in blood lead levels. This relationship was only evident in infants whose mothers had Pb-B levels of ~8 µg/dl or higher.

C. DOSE-EFFECT/DOSE-RESPONSE RELATIONSHIPS FOR PEDIATRIC LEAD EXPOSURE

In this section, information from the two previous subsections is integrated to give a quantitative picture of the relationship between adverse outcomes and Pb-B levels in children. Outcomes are measured as either categorical variables or continuous variables, conventionally termed responses and effects, respectively. Thus, one may refer to either dose-response or dose-effect relationships, depending on whether the outcome is discrete or continuous.

Table IV-3 summarizes lowest observed effect levels (LOELs) for a variety of important adverse health effects in children, based on a critical evaluation and interpretation of these findings by U.S. EPA (1986a), with updating. As shown in the table, the severity of effects increases as lead exposure levels increase. However, a constellation of effects, including alterations in neurobehavioral development and electrophysiological function, disturbances in heme biosynthesis, and deficits in growth and maturation, both prenatally and later in childhood, is evident at blood lead levels of 10 to 15 μg/dl, and possibly lower (U.S. EPA, 1986a). Some recent work also suggests that auditory acuity is reduced at these levels as well.

At levels still below 20 μg/dl, erythrocyte protoporphyrin is elevated, and disturbances in 1,25-$(OH)_2$-vitamin D and early signs of impaired erythropoietic pyrimidine metabolism are evident. Cross-sectional studies reveal IQ deficits at Pb-B levels starting below 25 μg/dl and at progressively higher levels as well.

A detailed dose-response relationship in a population of children has been described only for EP elevation. Using probit techniques, Piomelli et al. (1982) have reported the EP dose-response relationship depicted in Figure IV-3. This plot represents response at both 1 and 2 standard deviations as Pb-B rises. The impression to be gained from Table IV-3 is that the Pb-B threshold level of 15 to 25 μg/dl is already associated with the onset of a number of early biological changes.

This report provides population estimations in various chapters in terms of three Pb-B ceilings: 25 μg/dl (based on CDC, 1985), 20 μg/dl (based on WHO, 1986), and 15 μg/dl (based on U.S. EPA, 1986a). Selecting these particular values for the purposes of this report does not imply that lower levels are safe.

144

TABLE IV-3. LOWEST OBSERVABLE EFFECT LEVEL (Pb-B) FOR EFFECTS IN CHILDREN[a]

Lowest Effect Pb-B (μg/dl)	Neurological Effects	Heme Synthesis Effects	Other Effects
10-15 (prenatal & postnatal)	Deficits in neurobehavioral development (Bayley and McCarthy Scales); electrophysiological changes	ALA-D inhibition	Reduced gestational age and weight at birth; reduced size up to age 7-8 years
15-20		EP elevation	Impaired vitamin D metabolism; Py-5-N inhibition
<25	Lower IQ, slower reaction time (studied cross-sectionally)		
30	Slowed nerve conduction velocity		
40		Reduced hemoglobin; elevated CP and ALA-U	
70	Peripheral neuropathies	Frank anemia	
80-100	Encephalopathy		Colic, other GI effects; kidney effects

[a] Adapted from U.S. EPA (1986a), with updating.

D. PERSISTENCE OF ADVERSE HEALTH EFFECTS FROM LEAD EXPOSURE IN YOUNG CHILDREN

For this topic, these questions should be considered:

(1) What is the time frame to be defined under the term persistent? Does it include only childhood or the entire life span?

(2) Are the persistent effects to be compared with societal or clinical values and judgments that encompass optimal psychological and physical well-being, or simply the absence of overt disease?

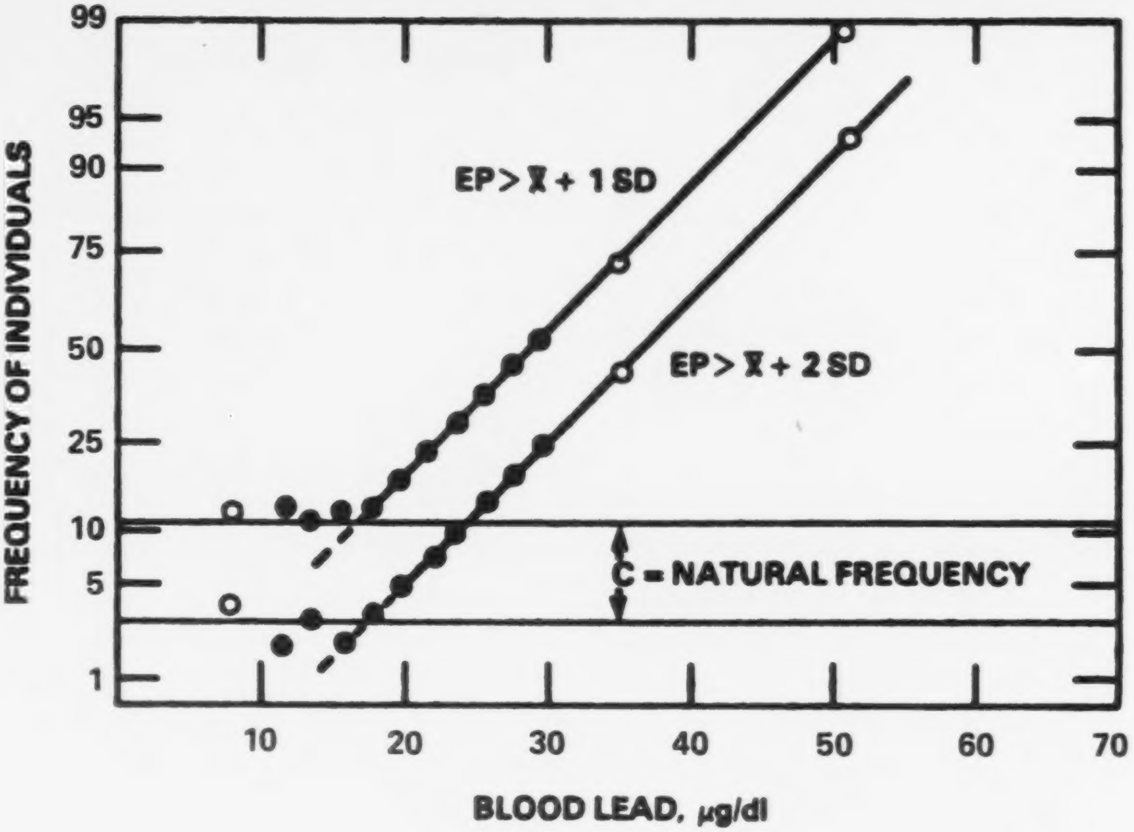


Figure IV-3. Dose-response for evaluation of EP as a function of blood lead level using probit analysis. Geometric mean plus 1 S.D. = 33 µg/dl; geometric mean plus 2 S.D. = 53 µg/dl.

Source: Piomelli et al. (1982).

(3) If lead-associated effects induced in childhood resolve themselves beyond childhood, what evidence remains that there are no deficits in overall development associated with these effects? For example, even if IQ decrements are present only in early childhood, what other more permanent deficits may be acquired in terms of emotional development, social interactions, and other facets of human development?

(4) Might one have to reconcile de facto meanings for the inherent reversibility or irreversibility of certain effects? As noted earlier, even in the face of biological reversibility there may be socioeconomic irreversibility of lead exposure and hence potential adverse health effects. In addition, there may be persistence of internal lead exposure well beyond early childhood in individuals burdened by bone lead accumulations that are later released into other compartments of the body.

It is generally considered that lead-induced lesions of the central nervous system are largely irreversible (American Academy of Pediatrics, 1987). Table IV-4 tabulates various lead-induced effects due to prenatal or postnatal exposure, with comments about the persistence of such effects. These assessments spring from both cross-sectional and prospective epidemiologic studies, and provide multiple indications of persisting, long-term health effects in children. However, firm conclusions about the persistence and ultimate impact of such effects are difficult to state at present because of the limited time spans over which children have thus far been studied. A more definitive assessment of the persistence issue will require the continuing examination of children in prospective studies.

As noted by Grant and Davis (1987), ontogeny is characterized both by its plasticity and by its sequential dependency. Developing organisms may be able to compensate for certain deficiencies, if they occur early enough in the maturation of the individual. For example, children often show catch-up growth spurts. Thus, it is possible that early developmental lags, particularly those that are somewhat subtle, could "disappear" at later ages. But it is also important to note that, even if a lead-induced lag in cognitive or physical development were no longer detectable later (which depends very much on the sensitivity of available measurement methods), this would not necessarily imply that the earlier impairment was without consequence. Research in developmental and physiological psychology has clearly shown that the actualization of behavioral capabilities requires appropriate periods of functional neural activity for proper development. Thus, even transient or, in themselves, reversible deficits during early development may have potentially serious and long-lasting sequelae. Moreover, secondary effects of early developmental perturbations need not be strictly sequential. Given the complex interactions that figure into the psychosocial development of children, attempts to compensate for lead-induced deficits in one area of a child's development may affect other areas of development.

Of particular relevance to the question of persisting effects of lead on neurobehavioral function is a large body of experimental animal research that demonstrates deficits in various aspects of behavior for several years after experimentally controlled lead exposure has been terminated. These effects have been reliably and consistently found in nonhuman primates as well as other species. Discussion of these studies is beyond the scope of this report;

TABLE IV-4. RELATIVE PERSISTENCE OF ADVERSE HEALTH EFFECTS IN INFANTS AND CHILDREN

Adverse Effect	Length of Study Period(s)	Comments
Reduced gestational age and birth weight	Birth onwards, developmental deficits. Pregnant mothers enrolled prior to delivery, offspring followed up to 24 months postnatally, thus far.	Major predictors for persisting, later developmental problems.
Deficits in Bayley Mental Development Index	Up to 24 months thus far.	Early neurobehavioral tests assess functional health of nervous system in infants.
Preliminary indications of deficits in McCarthy Scales performance at 5 years of age	Relationship observed over 3-year interval thus far.	Effect related to postnatal exposure at 2 years of age.
IQ deficits in school-age children and other measures	With higher Pb-B levels, IQ deficits persist. Low levels show several years of persistence w/Pb-dentine; may not be detectable with Pb-B. Reaction time effects appear to persist up to 6 yrs post-exposure, using Pb-tooth as index.	Any persistence in IQ deficits carries risks for other psychosocial effects. Good evidence for persistence of cognitive deficits stems from nonhuman primate data showing related but irreversible impairment of learning acquisition. Ongoing prospective studies will provide key to many questions.
Neurophysiological disturbances	Five years after most Pb-vulnerable period, effects remain on CNS sensory pathways depending on conditioning paradigm employed.	Passive conditioning stimulation approaches show persistence up to 2 years; no persistence at 5 years. Active conditioning task testing not done originally.
EP elevations	Elevations persist with both external exposure and endogenous (bone) lead release.	Cascade of effects from body heme pool disturbances (see Figure IV-2) include neurological development.

[a] Source: U.S. EPA (1986a) and Davis and Svendsgard (1987), with updating.

the interested reader is referred to a comprehensive review of this work in Air Quality Criteria for Lead (U.S. EPA, 1986a, Chapter 12).

We have a rather good understanding about the persistence of the effects induced in the heme biosynthesis pathway when exposure is maintained. In populations at high risk for lead exposure, EP elevation is a chronic problem (see U.S. EPA, 1986a). Furthermore, elevation of EP can persist beyond early childhood. Persistence in EP elevation is particularly likely in cases where Pb-B levels remain elevated because of resorbable bone lead. One need only examine Figure IV-2 to realize the potential for extended persistence of effects in a myriad of other systems, when the heme pool in the body remains disturbed.

In summary, then, and in response to the relevant language in Section 118(f), we can state that various adverse effects of lead do persist, or can potentially persist, over extended time periods. Furthermore, such persistence need not be of long duration to have implications for future deleterious effects on physical and psychosocial development.

V. EXAMINATION OF NUMBERS OF LEAD-EXPOSED CHILDREN BY AREAS OF THE UNITED STATES

Section 118(f)(1)(A) of the Superfund reauthorization legislation calls for an estimate of the numbers of children exposed to lead levels high enough to cause adverse health effects. The numbers are to be arrayed by Standard Metropolitan Statistical Area (SMSA) or some other geographical unit.

As noted in Chapters II and IV, exposure can be defined as the level of lead in whole blood (Pb-B), and "exposure sufficient to cause adverse health effects" is defined as a Pb-B level at or above which such effects are manifested. The specific reference Pb-B values, which we use to define adverse health effects, were presented in Chapter IV. In Section 118(f)(1)(A), lead exposures are defined in terms of adverse health effects as we now understand them. Since the levels of acceptable lead exposure in terms of blood lead concentrations are continuously being lowered in response to new evidence of low-level lead effects, these indices may decline even further. The reader should bear this in mind when examining findings in this and other chapters in the report. Low-level lead effects occur across a range of Pb-B values, that is, 10, 15, 20, and 25 µg/dl, and these ranges are considered where it is appropriate.

The obvious response to the Congressional directive (i.e., tabulating Pb-B levels gathered for each child in each geographical unit) is not possible at present. Such information does not exist at this time. A response would be easier to achieve if current data representative of the prevalences of Pb-B levels in highly defined geographical areas and socioeconomic/demographic strata within those areas were available, but here also, information is not readily available. We then are required to base estimates on whatever information is available.

To evaluate lead exposure, i.e., elevated Pb-B levels in populations, we used "enumerations" or actual physical counts of subjects, or else the most reliable derivations of numbers based on estimates for groups. For estimates, we can employ Pb-B prevalence modelling in tandem with population strata. When combined these data give estimates of the number of subjects that have Pb-B levels above different Pb-B criterion values, whatever these criterion values actually mean in terms of toxicity or toxicity risk. As a simple example, we can determine the actual numbers of children stratified by certain socio-economic/demographic categories at a national or other level, based on census enumerations. If a second data set tells us that children in one of the above categories have a prevalence of 25% for Pb-B levels above, say, 15 µg/dl, then a simple multiplication of the census number by the prevalence fraction gives us the number of children estimated to be at Pb-B levels above 15 µg/dl in this stratum.

For enumerations, we will present the number of children actually identified with elevated Pb-B levels in various screening programs. Since these programs were conducted in discrete geographical locations, their results can be included as a response to the Congressional directive.

Estimates and enumerations appear in Sections A and B, respectively. In working with available data, we found that we could also quantify the U.S. Census-enumerated children in each of the 318 SMSAs who may have been exposed to lead through leaded paint in their houses or housing-related environment. Although the report presented in Section C for these children differs from the reports in Sections A and B, insofar as we cannot assess actual exposure through their Pb-B levels, they represent actual, essentially metropolitan depictions of child exposure risk for leaded paint-related exposure as of 1980. In their case therefore, we are combining a geographic variable with a source variable. Section D summarizes the various exposure examination methodologies and their respective results.

A. ESTIMATED NUMBERS OF LEAD-EXPOSED CHILDREN IN SMSAs BY SELECTED BLOOD LEAD CRITERION VALUES

In this section, we estimate the number of children living in the SMSA segment of the country who have been exposed to lead at levels large enough to affect their health. We focus on the risk of urbanized populations in the United States, rather than the entire population, since Congress judged this population segment to be of concern. To enumerate this urbanized population, as described below, data was used on children in the SMSAs as defined by the

U.S. Bureau of the Census in 1980. The updated and more recent definitions, Metropolitan Statistical Areas (MSAs) and Consolidated Metropolitan Statistical Area (CMSA), could not be used for methodological reasons.

We selected 1984 as the reference year for estimating the number of young children at or above selected Pb-B levels because 1984 was the most recent year having all the required counts available to us. The child population data for 1984 give the potential number of children who may be at risk for adverse health effects from exposure to lead and helps us to assess the size of these risk groups across SMSAs. However, this method does not provide a quantitative assessment of the number of children with elevated lead exposure.

To estimate the number of children who will have elevated lead exposure, we need data that provide prevalences for each of the demographic and socioeconomic strata within the child population, which can be applied nationwide. Such variable prevalences are needed because the exposure levels are known to vary significantly and substantially between strata, and an "average national prevalence" would not estimate accurately the number of children at risk.

Findings from the second National Health and Nutrition Examination Survey (NHANES II) give such prevalences since they provide a nationwide picture of lead exposure in children and adults. The NHANES II data base has been used to estimate the number of children and other risk populations having Pb-B levels in the range covered in this report.

1. Estimation Strategies and Methods

Three basic steps were involved in the estimates: (1) enumerating the total number of children in each SMSA and allocating them to the selected strata as defined by age, race, income, and, where possible, urbanization categories to match the strata employed in the NHANES II analysis; (2) summing specific SMSA strata populations to obtain national totals for each stratum; (3) multiplying each stratum population (national total) by the prevalences for the three selected criterion Pb-B levels, after adjusting prevalences from their 1978 levels to 1984 levels, to account for the reduction of lead in gasoline.

The NHANES II survey reported prevalence of Pb-B values as a function of socioeconomic/demographic and ethnic strata of children across the nation, but not for specific geographic population clusters, such as SMSAs or cities, or legal entities, such as municipalities, counties, or states. For example, we can consider white or black children residing inside the central city of an SMSA with a population of over 1 million who were aged 0.5 to 5 years and in a family with an annual income of less than $6,000, but we cannot consider children in the Springfield, IL, SMSA. The NHANES II data show that Pb-B levels are more similar in children who share the characteristics of a particular stratum across the country than children of different strata who live in the same local area.

To apply the NHANES II prevalences in a statistically valid manner, we sorted out the SMSA child populations as provided by the 1980 Census tapes into the population strata used by NHANES II. However, since we were interested in child populations in 1984, a more recent year, we proceeded as follows.

The actual counts from the 1980 Census are available on data tapes from the U.S. Bureau of the Census. However, these tapes contain selected parts of the information. We used a tape called "Public Use Microdata Samples," which contains the counts for all the 318 SMSAs defined for 1980. To present users with a manageable data tape, the U.S. Census Bureau omitted some of the detail for some of the data units, creating several problems for us. (1) The population figures, originally exact counts, had been rounded off to the nearest hundred. This became a problem when working with the data for black children since the total number of black children in many SMSAs is quite small, and frequently their distribution among the required socioeconomic strata results in totals below 100 in a given stratum. In these cases, the tape showed zero as the number in one or more of the race/age/urban status/family income categories. This left us without a basis for assigning proportions of children into strata, and consequently we could not estimate the numbers of children at selected criterion values of Pb-B for these particular cases. This proved to be a particular problem in SMSAs with populations of less than 200,000. This particular problem of allocation is less significant when SMSAs are viewed collectively and not individually, as we had originally hoped to be able to do.

(2) In the available data tape, 34 of the SMSAs were merged into 17 pairs; making it impossible to separate the data for the pairs. These 17 pairs are listed in the introduction to Appendix C. The selection of the pairs appears to control for geographic location, population size, and other characteristics. Examples of such pairing are Bangor and Lewiston-Auburn, ME, and Midland and San Angelo, TX. These paired SMSAs account for all except one SMSA with populations below 100,000.

154

(3) The population in some SMSAs could not be separated by the urban status, "Inside Central City," and "Outside Central City." One SMSA did not contain any population center meeting the census definition of "Central City", and the tape failed to distinguish central city residents from all others for the 17 merged pairs. This necessitated presentation of our findings for three SMSA sets of estimates and also required reanalyzing NHANES II data to accommodate this problem since originally the NHANES II data available required stratification by these variables.

The data we present is limited to young white and black children because the NHANES II survey did not include enough children of Hispanic and "other race" origins for calculating reliable prevalences. NHANES III plans to correct this but the field work is not scheduled until 1988.

Tabulating the tapes for 1980 gave us the distribution of young children in two race and two age categories (0.5 to 2 years and 3 to 5 years) by urban status and family income groups. A further stratification variable, the size of an SMSA's total population as either over or under 1 million, was also known. We applied these 1980 distributions to the number of children established as the 1984 child population 0.5 to 2 years and 3 to 5 years for the two races (see below for construction of 1984 populations. The two age bands were merged later when prevalences were used.) The 1982 recession and its effect on the economic status of the population (family income) could not be taken into account, since there was no method of establishing its impact on each individual SMSA. When interpreting findings, remember that the actual 1984 distribution by income contained larger proportions of the population in the lower income categories. This is of crucial significance since income is an independent variable in the distribution of Pb-B levels, with the lower income segments of the population constituting a larger proportion of all groups with elevated Pb-B levels than do higher income groups.

The most recent natality statistics available were for 1984, and we compiled the white and black child population 0.5 to 2 years and 3 to 5 years as of 1984 by the following steps for each SMSA. We established the actual census counts of children up to 2-years old in 1980 and added to these counts the resident live births minus infant deaths for 1981. This gave us the age group 3 to 5 years. Natality and infant death data were obtained for 1982, 1983, and 1984. The numbers for 1984 were divided in half to yield the 0.5- to 1-year olds for the 0.5- to 2-years-old group. The census counts and natality and infant mortality data were all available by race. The Division of Vital Statistics of the National Center for Health Statistics supplied the published data for 1981 (USDHHS, 1985a, 1986) and printouts of the as yet unpublished data for the more recent years.

Further stratifications (other than race, age, and size of SMSA) that were employed by NHANES II were accomplished by applying the distributions for Inside/Outside Central City and three categories of family income as found in the 1980 Census counts for children then 0.5- to 2- and 3- to 5-years old. For example, in a given SMSA, 75% of the black children 0.5- to 2-years old in 1980 lived inside the central city and 25% outside. We applied these percentages to the black children 0.5- to 2-years old in 1984 in that SMSA. For the 75% living inside the central city in the 1980 Census counts, we established the percentages for each of the three family income categories and applied those rates to the 75% of the black children 0.5- to 2-years old in 1984. We repeated this process for the 25% of the children outside the central city. Using this process, we could establish the 1984 child population strata by NHANES II characteristics in each SMSA. We established 24 strata for those SMSAs where the data user tape included details on Inside/Outside Central City residential status. Since the SMSAs population size, over or under 1 million, was also a stratification variable in NHANES II, we established 48 strata for the two larger types of SMSAs. As noted earlier, the data tape did not permit Inside/Outside Central City stratification for smaller and paired SMSAs, and we could establish only 12 strata for these SMSAs. A total of 60 strata resulted. When the two age bands were merged, 30 strata resulted for estimating numbers at the selected criterion levels of Pb-B.

The NHANES II analyses originally summarized and published Pb-B levels that did not include all the levels of interest to this report, namely: >15, >20, and >25 μg/dl. Chapter IV discussed the rationale for selecting these levels. Further, the original NHANES II prevalences were calculated for 1978, and applying those rates to 1984 populations would overestimate children at risk. These 1978 prevalences are no longer accurate because total lead burdens in the environment have been reduced primarily by the decreases of lead in gasoline and to some extent in food. The NHANES II data shows the impact of the gasoline lead phasedown over 1976-1980. Because the amount of lead in the environment continues to decrease, a method for projecting prevalences from 1978 to our reference year of 1984 was necessary. Therefore, EPA's Office of Policy

Analysis (J. Schwartz and H. Pitcher) performed a statistical procedure called logistic regression analysis to update estimates of prevalences to 1984 and produce prevalences at the selected criterion values of >15, >20, and >25 μg/dl for SMSA populations of children in the required strata. A detailed discussion of this logistic regression/projection methodology is presented in the second part of Appendix G.

2. Results

Tables V-1 and V-2 give estimated prevalences of Pb-B levels in children above three selected levels; 15, 20, and 25 μg/dl within the strata. Tables V-1 and V-2, respectively, show these rates for "Inside Central City" and "Outside Central City" categories. Shown are three family income levels to indicate relative poverty, and each income category has two classifications by race. Each of these 6 strata is further divided into "SMSA with population ≥1 million" and "SMSA with population <1 million." In 122 SMSAs, we had 12 strata available to us, and each strata gave us three prevalence estimates.

TABLE V-1. PROJECTED PERCENTAGES OF CHILDREN 0.5-5 YEARS OLD ESTIMATED TO EXCEED SELECTED Pb-B CRITERION VALUES (μg/dl) BY FAMILY INCOME, RACE, AND URBAN STATUS,[a] WHO LIVE "INSIDE CENTRAL CITY" OF SMSAs, 1984

Family Income/ Race	>15 μg/dl		>20 μg/dl		>25 μg/dl	
	<1 M	≥1 M	<1 M	≥1 M	<1 M	≥1 M
<$6,000						
White	25.7	36.0	7.6	11.2	2.1	3.0
Black	55.5	67.8	22.8	30.8	7.7	10.6
$6,000-14,999						
White	15.2	22.9	4.0	6.1	1.1	1.5
Black	41.1	53.6	14.1	19.9	4.1	5.9
≥$15,000						
White	7.1	11.9	1.5	2.5	0.4	0.5
Black	26.6	38.2	6.8	10.4	1.5	2.2

[a] SMSA with population <1 million (<1 M) and SMSA with population ≥1 million (≥1 M).

TABLE V-2. PROJECTED PERCENTAGES OF CHILDREN 0.5-5 YEARS OLD ESTIMATED TO EXCEED SELECTED Pb-B CRITERION VALUES (μg/dl) BY FAMILY INCOME, RACE, AND URBAN STATUS,[a] WHO LIVE "OUTSIDE CENTRAL CITY" OF SMSAs, 1984

Family Income/ Race	>15 μg/dl		>20 μg/dl		>25 μg/dl	
	<1 M	≥1 M	<1 M	≥1 M	<1 M	≥1 M
<$6,000						
White	19.2	27.7	5.6	8.4	1.6	2.3
Black	45.9	57.8	17.9	24.5	6.1	8.4
$6,000-14,999						
White	10.9	16.8	2.9	4.5	0.8	1.2
Black	32.4	43.7	10.7	15.4	3.2	4.6
≥$15,000						
White	4.7	8.1	1.0	1.7	0.2	0.4
Black	19.5	28.9	4.9	7.6	1.1	1.7

[a] SMSA with population <1 million (<1 M) and SMSA with population ≥1 million (≥1 M).

Table V-3 shows the more limited number of strata and the relevant sets of prevalence estimates when Inside/Outside Central City status could not be ascertained. This applied to 196 SMSAs: 34 paired, 161 with populations below 200,000 (with very few exceptions), and Nassau-Suffolk, NY, with a population over 1 million but no central city.

The NHANES II Pb-B levels reported and used in calculating prevalences for criterion levels are based on Pb-B determinations for all cases--they are not influenced by initial erythrocyte protoporphyrin (EP) determinations and, for cases with elevated EP levels, subsequently selected Pb-B determinations.

In earlier analyses, we attempted to apply the national, urbanized composite prevalences to each of the SMSAs with the appropriate qualifications as to their reliability. This approach was attempted to best respond to the directive of Section 118(f) that asked for ranking of individual SMSAs. When the scientific community was reviewing this approach, however, problems were found with the level of permissible disaggregation due to the sample design of the NHANES II survey. For example, the national source-based differences that were implicit but not specific in the original analytic process could not be broken out and reassigned in disaggregation.

TABLE V-3. PROJECTED PERCENTAGES OF CHILDREN 0.5-5 YEARS OLD ESTIMATED TO EXCEED SELECTED Pb-B CRITERION VALUES BY FAMILY INCOME AND RACE WHO LIVE IN SMALL SMSAs,[a] 1984

Family Income/ Race	>15 µg/dl	>20 µg/dl	>25 µg/dl
<$6,000			
White	23.9	6.9	1.8
Black	56.5	22.9	7.4
$6,000-14,999			
White	13.2	3.4	0.9
Black	40.3	13.6	4.0
≥$15,000			
White	5.8	1.2	0.3
Black	25.4	6.3	1.4

[a] SMSAs with less than 1 million population.

In a second attempt to geographically specify lead exposure, projected prevalence estimates were calculated for the four major regions used in the original NHANES II survey: Northeast, Midwest, West, and South. However, statistical projection data could not be established for these regions. This was due in part to the small numbers of children with higher Pb-B/levels in some regions, such as the West. If these small numbers had been used to calculate prevalences in the region, some prevalences would have had unacceptable margins of estimating error. Consequently, in this report, we provide the updated Pb-B prevalence calculations for selected Pb-B levels and the nation's urbanized childhood lead-exposure status for each of the 30 socioeconomic/demographic strata described earlier.

Tables V-4, V-5, and V-6 present the results of applying the estimated prevalences of the 30 strata of children in all SMSAs. Table V-4 depicts children living inside central cities for the SMSAs where such division was possible, and Table V-5 shows the children outside central cities in these SMSAs. Table V-6 shows the findings for smaller and paired SMSA child populations. A partial summary of these three tables for children with Pb-B levels above 15 µg/dl is presented in Table V-7. Table V-8 presents overall summary data.

TABLE V-4. ESTIMATED NUMBERS OF CHILDREN, 0.5-5 YEARS OLD, WHO ARE PROJECTED TO EXCEED THREE LEVELS OF BLOOD LEAD (µg/dl) BY FAMILY INCOME AND RACE LIVING INSIDE CENTRAL CITIES IN SMSAs,[a,b] 1984

Family Income/ Race		SMSAs <1 M Pb-B µg/dl				SMSA ≥1 M Pb-B µg/dl		
	Number	>15	>20	>25	Number	>15	>20	>25
<$6,000								
White	130,900	33,600	9,900	2,700	259,400	93,400	29,000	7,800
Black	142,200	78,900	32,400	10,900	346,400	234,900	106,700	36,700
Total[c]	273,100	112,500	42,300	13,600	605,800	328,300	135,700	44,500
$6,000-14,999								
White	287,300	43,700	11,500	3,200	493,300	113,000	30,000	7,400
Black	138,900	57,100	19,600	5,700	345,000	184,900	68,700	20,400
Total[c]	426,200	100,800	31,100	8,900	838,300	297,900	98,700	27,800
≥$15,000								
White	648,500	46,000	9,700	2,600	1,046,800	124,600	26,200	5,200
Black	157,000	41,800	10,700	2,400	395,300	151,000	41,100	8,700
Total[c]	805,500	87,800	20,400	5,000	1,442,100	275,600	67,300	13,900
National Total[c]	1,504,800	301,100	93,800	27,500	2,886,200	901,800	301,700	86,200

[a] Data for SMSAs in this table permit separation of population residing Inside/Outside Central Cities.

[b] SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).

[c] Totals by addition, not estimation.

TABLE V-5. ESTIMATED NUMBERS OF CHILDREN, 0.5-5 YEARS OLD, WHO ARE PROJECTED TO EXCEED THREE LEVELS OF BLOOD LEAD (μg/dl) BY FAMILY INCOME AND RACE LIVING NOT INSIDE CENTRAL CITIES IN SMSAs,[a,b] 1984

Family Income/ Race		SMSAs <1 M Pb-B μg/dl				SMSA ≥1 M Pb-B μg/dl		
	Number	>15	>20	>25	Number	>15	>20	>25
<$6,000								
White	143,000	27,400	8,000	2,300	256,600	71,100	21,600	5,900
Black	32,300	14,800	5,800	2,000	77,200	44,600	18,900	6,500
Total[c]	175,300	42,200	13,800	4,300	333,800	115,700	40,500	12,400
$6,000-14,999								
White	428,200	46,700	12,400	3,400	716,500	120,400	32,200	8,600
Black	54,000	17,500	5,800	1,700	114,300	49,900	17,600	5,300
Total[c]	482,200	64,200	18,200	5,100	830,800	170,300	49,800	13,900
≥$15,000								
White	1,300,400	61,100	13,000	2,600	2,977,400	241,200	50,600	11,900
Black	73,700	14,400	3,600	800	222,800	64,400	16,900	3,800
Total[c]	1,374,100	75,500	16,600	3,400	3,200,200	305,600	67,500	15,700
National Total[c]	2,031,600	181,900	48,600	12,800	4,364,800	591,600	157,800	42,000

[a] Data for SMSAs in this table permit separation of population residing Inside/Outside Central Cities.

[b] SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).

[c] Totals by addition, not estimation.

TABLE V-6. ESTIMATED NUMBER OF CHILDREN 0.5-5 YEARS OLD, WHO ARE PROJECTED TO EXCEED THREE LEVELS OF BLOOD LEAD (μg/dl) BY FAMILY INCOME AND RACE IN SMALL SMSAs,[a] 1984

Family Income/ Race	Number	Pb-B μg/dl		
		>15	>20	>25
<$6,000				
White	249,700	59,700	17,200	4,500
Black	100,200	56,600	23,000	7,400
Total[b]	349,900	116,300	40,200	11,900
$6,000-14,999				
White	741,000	97,800	25,200	6,700
Black	141,100	56,900	19,200	5,600
Total[b]	882,100	154,700	44,400	12,300
≥$15,000				
White	1,670,800	96,900	20,000	5,000
Black	143,000	36,300	9,000	2,000
Total[b]	1,813,800	133,200	29,000	7,000
Unstratified				
Total	6,800	*	*	*
National Total[b]	3,052,600	404,200	113,600	31,200

[a] SMSAs with less than 1 million population except Nassau-Suffolk, NY, which has more than 1 million but no Central City.

[b] Totals by addition, not estimation.

*No estimates possible.

In Table V-8, the overall findings show a 1984 child population of about 13,840,000 living in SMSAs. Of these, 2,381,000 are expected to have Pb-B levels above 15 μg/dl, indicating that about 17% of the target population is at risk for adverse health impacts from lead exposure. About 5% of the children would be expected to have Pb-B levels above 20 μg/dl and about 1.5% would have Pb-B levels above 25 μg/dl.

Before discussing these findings, their limitations as "national totals" must be emphasized. These limitations include both underestimates and overestimates due to the methodologies employed. These estimation uncertainties may

TABLE V-7. ESTIMATED NUMBERS OF CHILDREN, 0.5-5 YEARS OLD, WHO ARE PROJECTED TO EXCEED 15 μg/dl Pb-B, BY FAMILY INCOME AND RACE IN ALL SMSAs, 1984

Family Income/ Race	Population Base	Inside Central City[a]		Not Inside Central City[a]		Small SMSAs[b]	Total
		<1 M	≥1 M	<1 M	≥1 M		
<$6,000							
White	1,039,600	33,600	93,400	27,400	71,100	59,700	285,200
Black	698,300	78,900	234,900	14,800	44,600	56,600	429,800
Total[c]	1,737,900	112,500	328,300	42,200	115,700	116,300	715,000
$6,000-14,999							
White	2,666,300	43,700	113,000	46,700	120,400	97,800	421,600
Black	793,300	57,100	184,900	17,500	49,900	56,900	366,300
Total[c]	3,459,600	100,800	297,900	64,200	170,300	154,700	787,900
≥$15,000							
White	7,643,900	46,000	124,600	61,100	241,200	96,900	569,800
Black	991,800	41,800	151,000	14,400	64,400	36,300	307,900
Total[c]	8,635,700	87,800	275,600	75,500	305,600	133,200	877,700
National Total[c]	13,840,000[d]	301,100	901,800	181,900	591,600	404,200	2,380,600

[a] SMSAs with total population less than 1 million (<1 M) and SMSAs with total population of 1 million or more (≥1 M).

[b] SMSAs with less than 1 million population except Nassau-Suffolk, NY, which has more than 1 million but no Central City.

[c] Totals by addition, not estimation.

[d] Includes 6,800 children from small SMSAs who could not be stratified by family income.

TABLE V-8. SUMMARY OF ESTIMATED NUMBERS OF CHILDREN 0.5-5 YEARS OLD IN ALL SMSAs, WHO ARE PROJECTED TO EXCEED SELECTED LEVELS OF BLOOD LEAD, BY URBAN STATUS, 1984

Characteristic	Population Base	Blood Lead Level (µg/dl)		
		>15	>20	>25
In SMSAs >1,000,000	7,251,000	1,493,400	459,500	128,200
In Central City	2,886,200	901,800	301,700	86,200
Not In Central City	4,364,800	591,600	157,800	42,000
In SMSAs <1,000,000	3,536,400	483,000	142,400	40,300
In Central City	1,504,800	301,100	93,800	27,500
Not In Central City	2,031,600	181,900	48,600	12,800
In Small SMSAs	3,052,600[a]	404,200	113,600	31,200
National Total	13,840,000	2,380,600	715,500	200,700

[a] Total includes 6,800 children who could not be stratified by income and were not included in estimates for three Pb-B levels.

result from four sources. First, different numbers of children in each cell of the original NHANES II data base introduce different levels of precision into the prevalences. Second, the logistic regression analysis only accounted for the reduction of lead from a single source, leaded gasoline. Any reduction due to lowered levels in foods was not accounted for. The reduced amount of lead in food among the 30 strata of children cannot be calculated with available data; therefore, it is impossible to factor this reduction for the strata into the logistic regression analysis.

Third, the total number of all U.S. lead-exposed children has been underestimated due to the exclusion of sizable national population segments. The calculations made did not include children of Hispanic and "All Other Races" origins who live in SMSAs. They were excluded since no reliable prevalences could be calculated for them. In a considerable number of SMSAs, particularly in the West and Southwest regions of the country, these children account for larger totals than black children. Although no complete data sets are available for children of Hispanic origin, nor for any other significantly large race/ethnic origin groups, it is reasonable to assume that the association between high Pb-B levels and poverty would hold for such groups. Cultural and other differences are still undefined in terms of Pb-B. Finally, it should be realized that birthrates in these race/ethnic groups are relatively high and consequently, these children will constitute an ever increasing proportion of the total child population in the future.

Fourth, children residing in SMSAs account for about 80% of the total child population. According to available data, children not residing in SMSAs exhibit lower prevalences than SMSA-based rates. However, the data base is not adequate for calculating meaningful stratification.

In summary, it is impossible to define precisely the various elements of overestimations and underestimations. Therefore, the estimates presented should be characterized conservatively as best estimates that can be based on available scientific data.

The findings summarized in Table V-8 indicate the extent of the problem, but they obscure insights into demographic/socioeconomic characteristics that have been associated with varying prevalences. These can be observed for one Pb-B level (15 μg/dl) as indicated in Table V-7. The tables showing the estimates of prevalences for the strata (Tables V-1, V-2, and V-3) show the expected negative association of socioeconomic status and Pb-B level. A positive association is found for density of population. Residence in the central cities and race also are associated with the variations in prevalence.

The most important finding, however, is that no strata of these children are totally exempt from risk of Pb-B levels high enough to represent a potentially adverse health impact. A numerically very large stratum of children, characterized by family income above the poverty level and predominantly white, is found to be of suburban residential status (Not in Central City). Although the estimated prevalences in these children are relatively low, estimates of those at risk should not be ignored when planning screening and case finding programs because such a large number of children are in the stratum. White children in the highest income group, living "Outside Inner City" and estimated to have Pb-B levels above 15 μg/dl totaled about 350,000 nationwide. (For the small SMSAs, we added half the estimated numbers of white high-income children with that Pb-B level.)

Table V-7 summarizes the distribution of children predicted to show Pb-B levels above 15 μg/dl by the strata for all SMSAs. The residential distribution of children is reflected in this table: black children are overrepresented in the poverty and low income strata as well as in the inner city areas of the SMSAs. But the ubiquity of the exposure to lead at >15 μg/dl is the striking finding. There are no strata of children totally free of this potential health risk, which holds true for higher Pb-B levels as well.

B. NUMBERS OF LEAD-EXPOSED CHILDREN BY COMMUNITY-BASED SCREENING PROGRAMS

In the previous section, the number of lead-exposed children was identified and categorized by socioeconomic-demographic variables and prevalence of selected Pb-B levels. In this section, we will discuss U.S. communities that have screening programs for identifying young children at risk. Elevated Pb-B level plus elevated erythrocyte protoporphyrin (EP) in blood is the measure used for assessing this risk. Because these screening programs are defined geographically as to city, county, or state, these programs and their locales come within the general meaning of the directives of Section 118(f)(1)(A) of SARA. In general, children living in these screening sites are considered to be at highest risk for lead exposure/toxicity, as we presently understand it.

This section briefly discusses the Second National Health and Nutrition Examination Survey (NHANES II) to compare with the lead screening efforts used over the years.

1. Lead-Screening Programs

Before examining in detail the results of the various lead exposure screening programs operating in U.S. communities it is useful to consider how the screening programs developed and their current status for interpreting the results of these programs. An additional overview of the U.S. screening process is also presented later, in the chapter dealing with exposure abatement and related issues. A comprehensive history of the public health aspects and operational characteristics was presented by Lin-Fu (1985a,b).

Screening activities were first mandated and supported by the 1971 Lead-Based Paint Poisoning Prevention Act, and the actual project started in Fiscal Year (FY) 1972. Shortly thereafter, the U.S. Centers for Disease Control (CDC) was given administrative responsibilities for these activities. By FY 1981, under CDC's guidance, screening in the United States had expanded to more than 60 programs and represented eight regions of the Department of Health and Human

Services (DHHS). Part of CDC's overall efforts included establishing a laboratory proficiency testing service for Pb-B and EP measurements in screening and other child medicine services. This was under the aegis of the Center for Environmental Health.

In FY 1982, screening and other grant programs were incorporated into the Maternal and Child Health (MCH) Block Grant Program and administered by the Maternal and Child Health Division, Bureau of Health Care and Delivery Assistance, Health Resources and Services Administration, DHHS. Although Farfel (1985) proposes that this change was attended by funding reductions, it is not possible to identify an actual reduction figure. As noted by Lin-Fu (1987), each state receiving the block grant portion of money determined its own priorities within the MCH programs, including lead screening. Furthermore, in many cases lead-screening costs are included in budgets for comprehensive pediatric services (Lin-Fu, 1987).

At present, the Association of State and Territorial Health Officials' (ASTHO's) administrative unit, the Public Health Foundation, is collecting data on childhood lead-poisoning prevention efforts from the various states; however, participation is voluntary. A number of states and constituent programs within the states have attempted to maintain the same level of effort that prevailed under CDC administration of the programs. In general, this is the case for the major programs in New York City, Chicago, Baltimore, and Massachusetts.

In FYs 1982 and 1983, the numbers of reporting states were 26 and 33, respectively. Has the number of programs decreased? This question cannot be answered very well without detailed canvassing. During the present administrative period, each state agency reports their results, and a given state may have more than one program unit as defined under the former CDC system. In December 1986, ATSDR canvassed reporting states and other jurisdictions; 41 program units responded with usable data. For all responding programs, the count was over 45 units. This tally included the MCH projects in Massachusetts and counted the rest of the state as one screening unit.

Screening data for different geographic areas have been tabulated. For several reasons, including continuity across time and differences in program characteristics, shown are: (1) data for the final year of the original program administered by CDC, FY 1981; (2) screening results from the programs under the

Maternal and Child Health Block Grant as reported under the ASTHO program (FY 1983); and (3) survey results gathered by ATSDR in December 1986. The ATSDR survey include results from agencies reporting by fiscal and calendar years, reported within 1985 or 1986 or both, and from independent programs within the various states.

Within each program period and between program periods, several factors have influenced, and continue to influence, screening results. (1) The screening risk classifications have changed since the results set forth in the following tables were gathered. These various classification schemes are shown in Tables V-9, V-10, and V-11. (2) Targeting high-risk populations has probably changed over the years. From FY 1972 to 1981, the strategies for screening populations, under CDC guidance, were uniform. The main goal was to screen groups of children in the community judged at high risk and having high prevalence rates for elevated Pb-B levels and for lead poisoning serious enough to warrant medical or public health action. Although this screening may give the number of children exposed in high-risk areas, it does not necessarily reflect the nationwide status of the lead problem. This screening was appropriate for the original purpose, that is, to concentrate attention on those children who most urgently need screening.

TABLE V-9. CDC LEAD SCREENING CLASSIFICATION SCHEME, 1978-1985[a]

Blood Lead	Erythrocyte Protoporphyrin (μg/dl Whole Blood)		
(μg/dl)	50-109	110-249	>250
30-49	II	III	III
50-69	III	III	IV
≥70	b	IV	IV

[a] Classification numbers increase with increased "toxicity" risk and need for diagnostic evaluation. A given class, e.g., Class III, can represent a combination of Pb-B/EP results. See CDC (1978) statement for more details.

[b] Not commonly encountered in screened populations.

TABLE V-10. CDC LEAD SCREENING CLASSIFICATION SCHEME AS OF JANUARY 1985, USING THE HEMATOFLUOROMETER[a]

Blood Lead (µg/dl)	Erythrocyte Protoporphyrin (µg/dl Whole Blood)[b]			
	<35	35-74	75-174	≥175
Not Done	I	-[c]	-[c]	-[c]
≤24	I	I[a]	I[a]	EPP[e]
25-49	I[b]	II	III	III
50-69	-[d]	III	III	IV
≥70	-[d]	-[d]	IV	IV

[a] Instrument for field measurement of EP, see CDC (1985) statement.

[b] Zinc protoporphyrin measured with hematofluorometer and free EP with chemical method.

[c] Requires a Pb-B measurement.

[d] Not usually observed.

[e] Erythropoietic protoporphyria (EPP), a genetic disease, is a possibility.

TABLE V-11. CDC LEAD SCREENING CLASSIFICATION SCHEME AS OF JANUARY 1985, USING CHEMICAL ANALYSIS OF EP[a]

Blood Lead (µg/dl)	Erythrocyte Protoporphyrin (µg/dl Whole Blood)[b]			
	<35	35-109	110-249	≥250
Not Done	I	-[c]	-[c]	-[c]
<24	I	I[a]	I[a]	EPP[e]
25-49	I[b]	II	III	III
50-69	-[d]	III	III	IV
≥70	-[d]	-[d]	IV	IV

[a] See CDC (1985) statement for more details.

[b] "Free" erythrocyte protoporphyrin measured by chemical method.

[c] Requires a Pb-B measurement.

[d] Not usually observed.

[e] Erythropoietic protoporphyria (EPP), a genetic disease, is a possibility.

Screening programs are now conducted with methods that tend to underreport the true screening prevalences of Pb-B levels. Since an EP level is the first step in assessing lead exposure in young children, children who have a "normal" EP level but an elevated Pb-B level will not be counted as a subject risking toxicity. The rate of these "false negatives" was reported to be considerable (see earlier quantitative discussion in Chapter II). Furthermore, the true rate may be even higher than when the prevalence of lead exposure is determined mainly at the time of clinic visits or the equivalent, compared with, for example, intensive, door-to-door canvassing.

Tables V-9, V-10, and V-11 show changes in risk classifications that consist of lowering the Pb-B level for the lowest category by 5 μg (from 30 to 25 μg/dl) and lowering the EP level in whole blood by 15 units (from 50 to 35). As noted elsewhere, these changes represent a trade-off between the lead levels the pediatric health community sees as harmful and the logistics of screening and the technical limits of the EP measuring methods routinely used. In other words, Pb-B levels below the CDC level of 25 μg/dl for whole blood should not necessarily be viewed as "safe."

In Table V-12, we have presented groups of children screened in the CDC program for FY 1981 and have given the number of children positive for lead toxicity based on the older action levels of 30 μg/dl Pb-B and 50 μg/dl EP. The numbers of responses currently defined as positive are separated further into two risk groups, Class II and combined Classes III and IV. Data in Table V-12 are as reported in the Morbidity and Mortality Weekly Report (CDC, 1982).

Table V-13 shows a summary of the more recent screening results that ASTHO collected from state health agencies. Twenty-seven state agencies provided numbers of young children screened for lead toxicity, but only 24 provided data on confirmed cases of toxicity (Public Health Foundation, 1986).

To determine the current scope and outcomes for various lead screening programs, ATSDR asked all state and known county or city screening units for information on the numbers screened, the numbers of confirmed lead toxicity cases, and the relevant time periods. Data from this ATSDR survey are in Table V-14. The survey period covers the time during which CDC distributed its January 1985 lead statement. Data in this table therefore represent numbers from some combination of the former and present screening classification schemes (For schemes see Tables V-9 through V-11.)

TABLE V-12. RESULTS OF SCREENING IN U.S. CHILDHOOD LEAD POISONING PROJECTS, FY 1981[a]

Program	Number Screened	Numbers of Children With Pb Toxicity		
		Total	Class II	Classes III & IV
United States	535,730	21,897	14,446	7,451
DHHS Region I	51,282	1,622	1,042	580
Bridgeport, CT	4,619	142	84	58
Waterbury, CT	2,856	64	41	23
Augusta, ME	3,546	20	10	10
Boston, MA	20,250	615	408	207
Lawrence, MA	6,552	404	300	104
Worcester, MA	6,026	110	75	35
Rhode Island	7,433	267	124	143
DHHS Region II	171,728	8,786	6,013	2,773
Atlantic City, NJ	1,151	111	49	62
Camden, NJ	3,657	134	74	60
East Orange, NJ	3,401	132	84	48
Elizabeth, NJ	284	17	11	6
Jersey City, NJ	3,880	437	284	153
Long Branch, NJ	1,117	31	25	6
Newark, NJ	8,500	1,201	882	319
Paterson, NJ	4,305	385	282	103
Plainfield, NJ	3,334	121	89	32
New Jersy (other programs)	615	146	75	71
Erie Co., NY	7,134	330	249	81
Monroe Co., NY	5,735	349	278	101
New York City	115,864[b]	5,010[b]	3,382	1,628
Onondaga Co, NY	7,305	217	151	66
Westchester Co., NY	5,446	135	98	37
DHHS Region III	84,195	3,722	2,422	1,301
Delaware	4,876	136	98	38
Washington, DC	12,183	205	141	64
Baltimore, MD	21,840	576	381	195
Allentown-Bethlehem, PA	1,456	12	9	3
Chester, PA	2,126	42	29	13
Philadelphia, PA	22,126	2,399	1,541	858
Wilkes-Barre, PA	2,091	50	36	14
Lynchburg, VA	1,352	30	20	10
Newport News, VA	2,968	57	31	26
Norfolk, VA	4,075	65	43	22
Portsmouth, VA	2,462	55	35	20
Richmond, VA	5,800	75	42	33
DHHS Region IV	47,631	614	412	202
Augusta, GA	2,793	45	32	13
Savannah-Chatham Co., GA	3,555	129	83	46

(continued on following page)

TABLE V-12. (continued)

Program	Number Screened	Numbers of Children With Pb Toxicity		
		Total	Class II	Classes III & IV
Louisville, KY	10,147	178	124	54
Cabarrus Co., NC	982	13	9	4
South Carolina	27,667	231	155	76
Memphis, TN	2,487	18	9	9
DHHS Region V	108,430	5,087	3,255	1,832
Chicago, IL	32,861	2,070	1,153	917
Kankakee, IL	2,468	56	40	16
Madison County, IL	2,288	105	66	39
Rockford, IL	2,341	30	18	12
Waukegan-Lake Co., IL	3,570	35	22	13
Illinois (other programs)	5,184	145	84	61
Ft. Wayne, IN	532	19	11	8
Detroit, MI	19,281	926	652	274
Grand Rapids, MI	688	19	15	4
Wayne Co., MI	1,818	75	55	20
St. Paul, MN	2,107	15	5	10
Akron, OH	4,637	149	144	5
Cincinnati, OH	9,085	191	119	72
Cleveland, OH	14,151	921	668	253
Beloit, WI	779	15	11	4
Milwaukee, WI	6,640	316	192	124
DHHS Region VI	48,944	571	366	205
Arkansas	12,976	177	106	71
Louisiana	18,022	41	25	16
New Orleans, LA	12,858	291	199	92
Houston, TX	5,088	62	36	26
DHHS Region VII	19,487	1,486	935	551
Cedar Rapids/Linn Co., IA	3,115	42	29	13
Davenport/Scott Co., IA	2,005	34	19	15
St. Louis, MO	11,231	1,323	829	494
Springfield, MO	484	18	8	10
Omaha, NE	2,652	69	50	19
DHHS Region IX	4,033	9	2	7
Alameda Co., CA	529	-	-	-
Los Angeles, CA	3,504	9	2	7

---

[a] Using CDC classification scheme of 1978 CDC statement (CDC, 1978). Tabular data from CDC's Mortality and Morbidity Weekly Report, October, 1982 (CDC, 1982). In the 1978 classification, toxicity risk begins with Pb-B ≥30 μg/dl and EP ≥50 μg/dl whole blood.

[b] Estimated figures.

TABLE V-13. LEAD POISONING SCREENING OF CHILDREN REPORTED BY 27 STATE AND LOCAL AGENCIES, FY 1983[a]

State or Other Agency	Number of Children Screened	Cases of Confirmed Pb Toxicity (%)
Arkansas	4,787	70[b] (1.5)
Connecticut	22,533	-
Delaware	5,187	145 (2.8)
Washington, DC	15,750	132 (0.8)
Idaho	380	8 (2.1)
Illinois	25,340	136 (0.5)
Indiana	1,265	1[b] (0.1)
Iowa	2,478	30 (1.2)
Kansas	2,642	29 (1.1)
Kentucky	7,416	105 (1.4)
Louisiana	51,804	269 (0.5)
Maryland	45,000[b]	348[b] (0.8)[b]
Massachusetts	130,000[b]	1,000[b] (0.8)[b]
Michigan	14,700	434 (3.0)
Minnesota	1,816	18 (1.0)
Missouri	11,778	1,278 (10.9)
Nebraska	2,090	39 (1.9)
New Jersey	35,534	-(1,930 evaluated)[c]
New York	160,960	4,205 (2.6)
North Carolina	14,000	80 (0.6)
Ohio	19,543	-(416 evaluated)[c]
Pennsylvania	19,994	450 (2.3)
Rhode Island	10,364	180 (1.7)
South Carolina	23,832	88 (0.4)
Virginia	8,401	84 (1.0)
West Virginia	449	1 (0.2)
Wisconsin	4,322[b]	187[b] (4.3)[b]
TOTAL	676,571	9,317 (1.6)

[a] Public Health Foundation (1986).

[b] Estimated by respondent.

[c] Only totals evaluated; actual number of positives were not provided.

The responses ATSDR received indicate that the results are derived mainly from the old CDC classification scheme if FY 1985 was employed. Other programs, reporting for a calendar year of screening, sent tabulations reflecting the change over to the new 1985 CDC classification scheme during 1985. This latter case is typified by the programs of St. Louis and New York City.

When using lower Pb-B and EP levels to classify toxicity, we might expect the numbers of positive lead toxicity cases to rise. Such a prediction, nationwide, can be made using the NHANES II data to predict Pb-B distribution

TABLE V-14. CURRENT (1985-1986) LEAD SCREENING ACTIVITIES REPORTED BY STATE AND LOCAL PROGRAMS TO ATSDR[a,b]

Agency program	Number of Children Screened (Year)	Cases of Confirmed Pb Toxicity (%)	
Delaware	5,818 (FY 85/July-June)	130	(2.2)
Washington, DC	17,000 (FY 85/Oct-Sept)	595	(3.5)
Georgia			
Augusta	2,960 (FY 85/Oct-Sept)	808/8,644	(9.0)[c]
Savannah	5,684 (FY 85/Oct-Sept)		
Illinois			
Chicago	37,409 (CY 85)[d]	693	(1.8)[e]
Indiana	3,770 (FY 85)	17	(0.5)
Iowa			
12 counties	2,143 (CY 85)	28	(1.3)
Scott County	897 (Jan-Nov 86)	9	(1.0)
Kansas			
Wyandotte	5,098 (CY 85)	16	(0.3)
Worcester	8,161 (FY 85)	71	(0.9)
	9,658 (FY 86)	72	(0.8)
Michigan			
Detroit	20,248 (CY 85)	371	(1.8)
	13,132 (Jan-Aug 86)	392	(3.0)
Minnesota			
Hennepin Co. (Minneapolis)	3,563 (Jan 85-June 86)	26	(0.7)
St. Paul	8,555 (CY 85)	64	(0.8)
	8,553 (Jan-Nov, 86)	41	(0.5)
Mississippi	3,628 (FY 86, Oct-Sept)	29	(0.8)
Missouri			
St. Louis City	12,308 (CY 85)[f]	1,356	(11.0)
	9,758 (Jan-Oct 86)	1,653	(16.0)
Nebraska			
Douglas Co.	3,167 (FY 86/Oct-Sept)	29	(0.8)
Maryland			
Baltimore	30,583 (CY 85)	504	(1.7)
Remainder of state	18,132 (CY 85)	46	(0.3)

(continued on following page)

TABLE V-14. (continued)

Agency program	Number of Children Screened (Year)	Cases of Confirmed Pb Toxicity (%)
Massachusetts		
Statewide	142,000 (FY 85)	1,531 (1.0)
	166,900 (FY 86)	1,011 (0.6)
Maternal and Child Health Projects		
Boston	29,925 (FY 85)	507 (1.7)
	29,356 (FY 86)	337 (1.2)
Holyoke	1,547 (FY 86)	23 (1.5)
Merrimac Valley	5,050 (FY 85)	177 (3.5)
	3,619 (FY 86)	42 (1.2)
North Shore	4,038 (FY 86)	46 (1.2)
Southeastern Massachusetts University	4,745 (FY 85)	54 (1.1)
	3,775 (FY 86)	35 (0.9)
Springfield	1,735 (FY 85)	34 (2.0)
	352 (FY 86)	3 (0.9)
New Hampshire	5,021 (FY 85/July-June)	24 (0.5)
	6,483 (FY 86/July-June)	46 (0.7)
New Jersey	58,080 (CY 85)	1,690 (2.9)
New York		
New York City[f]	206,467 (FY 85)	1,337 (0.7)
Bronx	44,501	288 (0.7)
Brooklyn	72,314	720 (1.0)
Manhattan	47,456	154 (0.3)
Queens	38,604	154 (0.4)
Richmond	3,256	24 (0.7)
(Unknown)	(356)	
North Carolina	15,567 (FY 85/Oct-Sept)	66 (0.4)
Pennsylvania	22,894 (FY 85)	631 (0.3)
Philadelphia	15,133	357 (2.3)
N.E. Philadelphia	983	8 (0.8)
Allegheny Co.	2,092	32 (1.6)
Harrisburg	2,026	101 (4.9)
Erie Co.	1,080	9 (0.8)
Rhode Island	14,640 (CY 85)	280 (2.0)
South Carolina	64,993 (FY 86)	920 (1.2)

(continued on following page)

TABLE V-14. (continued)

Agency program	Number of Children Screened (Year)	Cases of Confirmed Pb Toxicity (%)
Texas		
Dallas City	35,000 (Average for several years)	350-700[g] (1-2)[g]
Vermont	402 (Jan.-Aug. 1985)	1 (0.3)
TOTAL	785,285	11,739 (1.5)[h]

[a] FY 85 programs mainly using CDC 1978 statement classification scheme. Calendar Year 1985 (CY 85) programs use 1985 scheme in some cases.

[b] FY 86 and CY 86 summaries mainly use 1985 CDC scheme.

[c] Sum of the two cities.

[d] First screens only.

[e] 1985 CDC scheme starting July 1985.

[f] 1985 CDC scheme starting October 1985; confirmed cases refer to actually medically managed, not Class II-IV positives.

[g] Estimated.

[h] Includes upper estimates of cases for Dallas, TX: 700.

patterns in young children. The summary report of Mahaffey et al. (1982) shows that between 1975-1980 about 10% of black children 3 to 5 years old had a Pb-B level of 30 µg/dl or above, whereas the corresponding figure for the 20-29 µg/dl Pb-B fraction of the distribution was 43.3%. The mean and median Pb-B values for this particular part of the population were 20.8 and 20.0 µg/dl, respectively. As discussed extensively by U.S. EPA (1985, 1986a), this distribution has shifted downward in children as a group and will continue to do so for some time. The magnitude of the shift in inner-city children, one of the target groups in screening, however, would probably be smaller than for U.S. children as a whole, which can be attributed to the disproportionately higher concentrations of lead in paint and related dusts in the environment of these children.

The changes in prevalence, which resulted from lowering the Pb-B and EP levels used for screening (CDC, 1985), can theoretically be seen in a program with stable protocols and targeting criteria. Fortunately, changes in lead toxicity rates were assessed in New York City for the last quarter of 1985 relative to the last quarter of 1984.

In Table V-15, for the October-December quarter of 1985, the number of New York City lead toxicity cases using the previous classification of CDC is compared with the number found using the new classification (Bureau of Lead Poisoning Control, Department of Health, City of New York, 1985). The numbers are only for the fourth quarter of 1985 because the new classifications were not used until October 1, 1985. When the previous classifications are used to compare the numbers for the fourth quarter of 1984 and 1985, the numbers of positives do not materially differ statistically. But when the new classification was used, the number of cases for the fourth quarter of 1985 increased 61.4% over the number for the fourth quarter of 1984 (502 cases compared with 311). Furthermore, the increase (the difference between 292 cases with the previous levels and 502 with the new--or 210 cases) is 42% of the cases for the fourth quarter of 1985.

TABLE V-15. IMPACT OF NEW CDC LEAD EXPOSURE SCREENING GUIDELINES ON THE NUMBER OF LEAD TOXICITY CASES IN NEW YORK CITY[a]

		1985 Cases	
	1984 Cases	Previous CDC Guidelines (% Change)[b]	New CDC Guidelines (% Change)[b]
October	132	119 (-9.8)	191 (+45.0)
November	99	95 (-4.0)	176 (+77.8)
December	70	78 (+11.3)	135 (+92.9)
TOTAL	311	292 (-6.0)	502 (+61.4)

[a] From: 1985 Annual Report, NYCDH, Bureau of Lead Poisoning Control.

[b] Relative to same quarter in 1984.

With the new CDC classification scheme, the number of lead toxicity cases should increase in communities having screening programs. The 1985 guidelines include reducing Pb-B, for the lowest classification, to a level near that prevailing in a significant fraction of urban children, ≥25 μg/dl. Changes in the New York City program (Table V-15) may or may not be typical of the impact, but programs with a large number of children with excessive lead exposure due to leaded paint and contaminated dust and soil are expected to increase.

When CDC redefined lead toxicity risk, the number of children potentially in the positive category significantly increased. Having a lower cutoff level may offset, to some extent, the decreases in Pb-B levels associated with reductions in some lead sources, such as gasoline and food, as noted earlier in this chapter.

From a broader perspective, do the various screening programs offer conclusive information about the extent of lead poisoning over the years? Clearly, the screening results given in the previous tables show markedly reduced numbers of cases of acute and subacute lead poisoning found in U.S. cities when compared to numbers, commonly due to leaded paint ingestion, found in the 1930s through the 1960s. Reducing the lead content of gasoline has helped lower the degree of chronic poisoning. In the past, sniffing leaded gasoline was an occasional source of subacute lead exposure, but as leaded gasoline is phased out (U.S. EPA, 1986a), the number of these cases should decrease.

The rate of chronic lead poisoning in young U.S. children appears to be decreasing moderately, but the absolute numbers of toxicity and the percentages of screenings that show toxicity still indicate a continuing problem. Table V-16 shows lead screening program results nationally and for two locations. The national figures for 1973 through 1981 are from CDC; for FY 1982 through FY 1984 from ASTHO (Public Health Foundation, 1986); and for 1985, from the December 1986 ATSDR surveys. Please note that the New York City rates of toxicity in Tables V-14 and V-16 refer to medically managed children and not total Class II-IV positives (see Table V-12 for such FY 1981 totals).

National screening results suggest a moderate decline in cases of lead toxicity over time. This is true even when complicating factors are considered--such as changes in protocols for laboratory quality assurance, new lead toxicity criteria, differences in levels of adherence to target population criteria, and the moderate decline in toxicity cases caused by reducing the lead content of gasoline. However, this table clearly shows that many children still suffer from lead toxicity. In St. Louis, for example, the number of lead toxicity cases has declined from the early 1970s, but the percentage of screening tests that are positive for toxicity is still unacceptable, and this percentage has not essentially changed for the past 6 to 7 years. For FY 1981, CDC figures of total positives (Table V-12) for New York City give a rate of 4.3%. The St. Louis program illustrates the difficulty in eliminating the prevalence of lead poisoning by communitywide intervention, even though the overall program efforts were effective over an extended time.

TABLE V-16. TEMPORAL VARIATION OF LEAD TOXICITY CASES IN SELECTED LEAD POISONING SCREENING PROGRAMS, 1973-1985[a,b]

	Pb Toxicity Cases (% Of Screened)		
Year	National	New York City[c]	St. Louis
1973	19,059 (6.4)	761 (0.6)	2,396 (32.3)
1974[d]	24,443 (5.4)	494 (0.4)	1,577 (27.0)
1975	30,343 (7.2)	1,559 (1.4)	2,530 (22.9)
1976	33,043 (8.1)	984 (1.0)	3,709 (28.0)
1977	28,072 (7.4)	652 (0.7)	3,519 (24.0)
1978[d]	26,734 (6.5)	802 (0.7)	2,080 (15.2)
1979	32,362 (6.8)	931 (0.8)	1,560 (12.5)
1980	25,293 (5.0)	976 (0.7)	1,422 (11.4)
1981	18,272 (3.6)	1,538 (1.2)	1,422 (12.4)
1982	10,114 (2.0)	1,259 (0.9)	1,278 (10.9)
1983	9,317 (1.6)	1,201 (0.8)	869 (7.6)
1984	5,035 (1.1)	979 (0.6)	1,066 (8.2)
1985[d]	11,739 (1.5)	1,337 (0.6)	1,356 (11.0)

[a] National screening figures given by CDC, 1973-1981 (CDC, 1982), by ASTHO for FYs 1982-1984, (Public Health Foundation, 1986) and by ATSDR survey for 1985 results.

[b] New York and St. Louis figures as provided to ATSDR by respondents.

[c] Positive cases refer to children hospitalized and given chelation therapy. The number is considerably less than total positive screens, above 40 µg/dl or all classes in 1978 classification scheme. See Table V-12 for total positive screening cases in New York City for FY 1981 (N = 5,010, 4.3%).

[d] Year in which CDC criteria for toxicity changed.

We also compared changes in exposure or toxicity evaluated in case studies with changes detailed in the usual screening program summaries. Chisolm et al. (1985) have pointed out that average Pb-B values in Baltimore may not be declining significantly among subjects at highest risk. For example, in 1956, the mean Pb-B level for a sizable group of children (N = 330) at highest risk was 43 µg/dl. In 1975, 19 years later, the mean Pb-B level for 155 children at highest risk was 38 µg/dl. These two means are both high and virtually indistinguishable.

A related question concerns whether the distribution of lead-intoxicated children among the three CDC risk categories changes over time independent of changes in the CDC classification schemes.

EPA, in its initial cost-benefit analysis of reducing lead in gasoline, examined quarterly data from CDC screening programs for the percentage of children with lead toxicity who were in Classes III and IV for the years 1977-1981 and found no change in the 20 quarters for these nationwide data (U.S. EPA, 1984).

The experience of the large Chicago Health Department program also suggests little change. Table V-17 shows the city's screening results for 1981 through 1985. Table V-17 shows little change over this time in the number of Chicago children in the highest and next-to-highest risk categories, (Classes IV and III, respectively). In Chicago, therefore, the distribution apparently is not shifting toward lower degrees of risk--that is, the percentages of children in Classes III and IV are not decreasing.

TABLE V-17. LEAD-SCREENING STATISTICS FOR THE CHICAGO DEPARTMENT OF HEALTH, 1981 TO 1985,[a] BY CDC CLASSIFICATION II, III, OR IV[b]

Year	Number First Screens	Initial Positive (IP)	II(% IP)	III(% IP)	IV(% IP)
1981	35,352	797	466 (58)	275 (35)	56 (7)
1982	34,499	875	563 (64)	324 (37)	51 (6)
1983	35,185	843	545 (65)	252 (30)	46 (5)
1984	35,961	837	549 (66)	238 (28)	50 (6)
1985	37,409	693	410 (59)	238 (34)	45 (6)

[a] Adapted from 1985 Report, Chicago Department of Health: Lead-Screening Statistics.

[b] See Table V-9 for description of classification scheme.

Finally, the numbers of reportedly "asymptomatic" children with high Pb-B levels entering the health care system for the first time should be examined. In particular, we need to assess the rates of these child admissions over the years and examine the corresponding mean Pb-B values in the groups of children admitted.

Such relationships have been described by Schneider and Lavenhar (1986), who examined the medical records of Newark, NJ, inner-city children hospitalized for treatment of lead toxicity. Table V-18 shows some of their results, including the rate of first hospital admissions per 10,000 urban children and the group mean Pb-B levels from 1972 through 1980. In Newark, NJ, the rate of child admissions for chelation therapy declined from 1972 until 1976, after which it increased significantly through 1980. This rise does not appear to be due to changes in chelation treatment criteria, and no movement downward in the Pb-B index signaled such a change. The rise in rate does coincide with declines in funding starting in 1976 and continuing through 1980. Because funds were

TABLE V-18. RATE OF FIRST HOSPITALIZATION AND ASSOCIATED **MEAN** Pb-B (μg/dl) IN "ASYMPTOMATIC" CHILDREN OF NEWARK, NJ, BY YEAR OF FIRST ADMISSION[a]

Year of Admission	Number of Children First Hospitalized	Rate[b]	Mean Pb-B (μg/dl)
1972	89	19.1	74.9
1973	81	18.0	76.6
1974	42	9.7	71.8
1975	40	9.6	69.6
1976	23	5.7	63.0
1977	28 (29)[c]	7.5	62.3
1978	32 (35)[c]	9.5	66.1
1979	42	11.9	63.7
1980	71 (72)[c]	21.4	64.7

[a] Adapted from Schneider and Lavenhar (1986).

[b] Per 10,000 Newark children.

[c] Number of children in the rate estimate.

decreased, the screening van and the public education program were eliminated and lead laboratory services were reduced. The Newark, NJ, experience is further discussed in the section on imperatives and strategies for abating lead poisoning and exposure (Chapter IX).

Are the results of state and local high-risk screening efforts duplicated in the nation in terms of present status and trends? We can answer this by examining results of the national NHANES II (1976-1980) study described earlier.

2. The NHANES II Study

Using the NHANES II statistical design and blood specimen collection program, analysis of EP and Pb-B values could be performed on a national level, thus adding new dimensions to the interpretation of results from screening programs. As Mahaffey et al. (1982) reported, the prevalence of Pb-B levels above 30 μg/dl (the action level for toxicity risk at the time of the NHANES II survey) in young children sampled by NHANES II was higher than predicted from screening data. When black children in low SES families in the central city were examined, very high prevalence rates were estimated. However, these rates seem to be reflected only in results of screening programs that target very similar pediatric populations, such as St. Louis and New York City. In Table V-19, NHANES II data for young children are summarized by race and age. The summary includes means, medians, and the Pb-B distribution at four levels. Within race, the two childhood age categories are not distinguishable as to means, medians, and distributions; but across race, higher levels observed in black children are statistically significant. Overall, urbanization and income are directly associated with lead exposure, especially among black children.

TABLE V-19. BLOOD LEAD LEVELS (μg/dl) IN U.S. CHILDREN BY RACE AND AGE, 1976-1980[a]

Race and Age	Mean Pb-B	Median Pb-B	Prevalences of Pb-B at μg/dl Levels of: <20	20-29	30-39	≥40
WHITE						
0.5-2 yr	15.0	14.0	80.2	17.3	2.2	0.3
3-5 yr	14.9	14.0	82.7	15.4	1.6	0.3
BLACK						
0.5-2 yr	20.9	19.0	50.5	34.2	13.0	2.3
3-5 yr	20.8	20.0	46.3	43.3	8.5	1.9

[a] Adapted from Mahaffey et al. (1982) and based on NHANES II data.

As noted earlier, the NHANES II data collected over the survey years showed a significant decline in Pb-B levels in children, which was strongly associated with the phasedown of lead in gasoline. A detailed discussion of this relationship is presented in the next chapter. Overall, the rate of the decline and related data suggested lower mean Pb-B levels for children in the 1980s. Results of the 1983 survey of Hispanic children, the "Hispanic NHANES," as yet available only as preliminary unpublished information, appear to agree with this suggested trend. Children in the highest risk categories--for example, inner-city black children in low SES families-- have more complex exposures, such as leaded paint and leaded paint weathering and chalking, than U.S. children as a whole. For this group, Pb-B levels may not be declining in the same way and at the same rate as for U.S. children as a whole.

C. RANKING OF CHILDREN WITH POTENTIAL EXPOSURE TO PAINT LEAD IN HOUSING

In the previous sections, estimates were given for the number of young white and black children in all SMSAs predicted to have Pb-B levels above selected criterion values as well as the findings from local screening programs. In these estimates, specific sources of lead were not considered.

Since the age of housing indicates the degree of exposure to lead in paint, we analyzed the distribution of children living in SMSAs by the age of their housing units. This discussion represents a combination of both area-based exposure and source-specific exposure, as presented in Chapter VI.

1. Strategies and Methods

As discussed in Chapter VI, age of housing in the U.S. may indicate general levels of exposure from leaded paint, e.g., the oldest housing has the highest lead concentration in its paint and the highest frequency (percentage) of leaded paint per age category.

In U.S. Census enumerations, as noted earlier, the larger SMSAs separated children living in central cities from those not in central cities. In addition, the data permitted grouping of the housing units into three categories: pre-1950, 1950-1969, and 1970-1980. The pre-1950 housing includes that portion of the housing stock that has the highest concentrations of lead in any leaded paint applied, i.e., 20 to 50% lead by weight. However, the paint supply used on pre-1940 housing, which had the very highest lead concentration, most likely carried over into the 1940s. During 1950-1969, lead concentrations were decreased, and post-1970 lead content was regulated and paint supplies with higher concentrations became exhausted.

The data for age of the housing units were available for all young children regardless of race/ethnic origin, and we were able to report this information for the total child populations, not just for white and black children. We have tabulated the census counts of children residing in old housing units for each SMSA, the percentage they represent, and have ranked the SMSAs by the number of children in pre-1950 housing in Table V-20. When several SMSAs were found to have the same number of children in pre-1950 housing, the percentage these children represented was used as a second ranking criterion and the SMSAs ordered from highest to lowest percentage (see #35 and #36 in Table V-20).

We also prepared detailed tabulations for each SMSA showing the same ranking of the SMSAs and including the numbers of children residing in the other two housing age groups, as well as the distributions for children "Inside Central City" and those "Outside Central City" when available. Appendix D contains this table. The paired SMSAs are shown separately at the end of the tables, since the pairs of SMSAs are reported as units and a similar distribution of age of housing had to be assumed for each of the pair.

The relationship of the children's distribution by the age of their housing and their economic status is of interest. According to popular wisdom and the definition of target populations for lead-screening programs, the poorest children are found in older housing. However, the findings shown in Tables V-20 and Appendix D located large numbers of children in old housing - more than would be accounted for by the population segment in poverty. Consequently, we further examined the relationship of income levels and residence by age of housing unit. Table V-21, an example of the resulting tabulations for each SMSA, shows an SMSA of over 1 million population. Appendices A, B, and C contain the tables for individual SMSAs in the three population size groups.

2. Results

Table V-20 summarizes all SMSAs ranked by the number of young children in pre-1950 housing. This ranking does not automatically correlate with the size of the SMSA populations nor does it correlate with the total SMSA population of young children. For example, Phoenix, AZ, was ranked 76th, and San Jose, CA, was ranked 85th, even though both are in the group of 38 SMSAs with total populations of over 1 million; nationally, Phoenix ranks 26th and San Jose ranks 30th in terms of total populations. It is not unexpected that the older population centers contain large proportions of children living in older housing. The chronology of urban and suburban growth varied enormously among SMSAs, as Appendix Table D shows. Some SMSAs grew most rapidly between 1950 and 1969, and others between 1970 and 1980. In general, for the older SMSAs, the children living in housing built from 1970-1980 represented a small percentage of those living in the central city. However, this finding is not true of SMSAs that have achieved maximum growth since the 1950s, as is apparent, for example, when comparing Buffalo, NY, and Houston, TX, ranked 17th and 20th, respectively.

TABLE V-20. RANKING OF 1980 CENSUS SMSAs BY NUMBER OF CHILDREN 0.5-5 YEARS OLD LIVING IN PRE-1950 HOUSING, AND TOTAL NUMBER OF YOUNG CHILDREN IN SMSA

		Children 0.5-5 Years		
Rank	SMSA	Number in pre-1950 housing	Percent of total in SMSA	Total Number in SMSA
1	New York, NY-NJ	422,800	60.1	703,500
2	Chicago, IL	271,500	43.2	628,800
3	Los Angeles-Long Beach, CA	225,700	33.5	628,800
4	Philadelphia, PA	172,500	46.4	371,800
5	Detroit, MI	141,900	37.8	375,800
6	Boston, MA	110,400	62.7	176,100
7	Newark, NJ	80,300	53.1	151,200
8	Cleveland, OH	75,100	49.1	153,000
9	San Francisco-Oakland, CA	74,800	32.3	231,500
10	Pittsburgh, PA	71,300	47.4	150,500
11	St. Louis, MO-IL	69,200	34.0	203,600
12	Minneapolis-St. Paul, MN-WI	60,000	32.2	186,600
13	Baltimore, MD	56,700	34.2	165,700
14	Milwaukee, WI	53,100	43.5	122,100
15	Nassau-Suffolk, NY	51,100	26.5	192,500
16	Washington, DC-MD-VA	48,900	20.9	234,400
17	Buffalo, NY	46,800	51.3	91,300
18	Cincinnati, OH-KY-IN	44,300	35.4	125,000
19	Dallas-Fort Worth, TX	40,900	15.3	267,400
20	Houston, TX	36,400	12.4	293,400
21	Rochester, NY	35,900	45.4	79,000
22	Jersey City, NJ	34,200	72.6	47,100
23	Albany-Schenectady-Troy, NY	34,000	54.3	62,600
24	Providence-Warwick-Pawtucket, RI-MA	33,300	53.3	62,500
25	Portland, OR	32,900	45.0	71,300
26	Toledo, OH-MI	32,100	45.0	71,300
27	Columbus, OH	31,500	30.4	103,600
28	Kansas City, MO-KA	31,500	28.0	112,300
29	New Orleans, LA	28,600	24.2	118,000
30	Seattle-Everett, WA	28,600	23.3	122,600
31	Indianapolis, IN	27,700	27.6	100,400
32	Riverside-San Bernardino-Ontario, CA	25,900	16.5	156,600
33	Denver-Boulder, CO	25,300	17.7	142,900
34	Syracuse, NY	24,700	45.7	54,000
35	Northeast Pennsylvania	24,400	53.0	46,000
36	Gary-Hammond-East Chicago, IN	24,400	37.3	65,500
37	San Diego, CA	23,900	15.4	154,800
38	San Antonio, TX	23,000	20.7	111,200
39	Akron, OH	22,900	40.2	56,900

(continued on following page)

TABLE V-20. (continued)

Rank	SMSA	Children 0.5-5 Years		
		Number in pre-1950 housing	Percent of total in SMSA	Total Number in SMSA
40	Dayton, OH	22,800	32.9	69,400
41	Hartford, CT	21,900	12.4	176,400
42	Atlanta, GA	21,900	12.4	176,400
43	Allentown-Bethlehem-Easton, PA-NJ	21,600	50.6	42,700
44	Grand Rapids, MI	21,000	37.0	56,800
45	Paterson-Clifton-Passaic, NJ	20,600	59.4	37,400
46	Salt Lake City-Ogden, UT	20,600	14.7	140,500
47	Louisville, KY-IN	19,800	24.7	80,100
48	Norfolk-VA Beach-Portsmouth, VA-NC	19,100	25.5	72,900
49	Flint, MI	19,000	36.1	52,600
50	Birmingham, AL	18,600	25.5	72,900
51	Youngstown-Warren, OH	18,000	40.1	44,900
52	Springfield-Chicopee-Holyoke, MA-CT	17,500	47.9	36,500
53	Lansing-East Lansing, MI	17,500	39.3	44,500
54	Fort Wayne, IN	16,500	43.8	37,700
55	Davenport-Rock Island-Moline, IA-IL	15,800	39.9	39,600
56	New Haven-West Haven, CT	15,600	47.6	32,800
57	Bridgeport, CT	14,800	53.0	27,900
58	Canton, OH	14,400	51.3	34,700
59	Sacramento, CA	14,100	16.4	86,100
60	Memphis, TN-AR-MS	14,100	16.4	86,100
61	Miami, FL	13,800	11.9	116,100
62	Wichita, KS	13,700	32.9	41,700
63	Anaheim-Santa Ana-Garden Grove, CA	13,500	8.5	159,600
64	Worcester, MA	13,400	51.0	26,300
65	Tacoma, WA	13,200	27.5	48,000
66	Omaha, NB-IA	13,100	26.3	49,800
67	Oklahoma City, OK	13,100	17.0	76,900
68	Tampa-St. Petersburg, FL	12,800	13.0	98,800
69	Duluth-Superior, MN-WI	12,600	50.4	25,000
70	South Bend, IN	12,600	45.8	27,500
71	Nashville-Davidson, TN	12,600	17.4	72,500
72	Peoria, IL	12,500	34.7	36,000
73	Fresno, CA	12,500	25.8	48,500
74	Wilmington, DE-NJ-MD	12,200	29.5	41,300
75	Utica-Rome, NY	12,000	46.7	25,700
76	Phoenix, AZ	12,000	9.4	127,900
77	Lawrence-Haverhill, MA-NH	11,900	54.3	21,900

(continued on following page)

TABLE V-20. (continued)

Rank	SMSA	Children 0.5-5 Years Number in pre-1950 housing	 Percent of total in SMSA	 Total Number in SMSA
78	Tulsa, OK	11,900	18.0	66,100
79	Huntington-Ashland, WV-KY-OH	11,400	40.4	28,500
80	Trenton, NJ	11,400	52.8	21,600
81	Jacksonville, FL	11,000	15.5	70,900
82	Binghamton, NY-PA	10,900	53.7	20,300
83	San Jose, CA	10,800	10.0	108,500
84	Newburgh-Middletown, NY	10,200	46.6	21,900
85	Erie, PA	10,000	44.1	22,700
86	Corpus Christi, TX	9,900	27.7	35,700
87	New Brunswick-Perth Amboy-Sayreville, NJ	9,800	22.9	42,800
88	Lexington-Fayette, KY	9,700	32.4	29,900
89	Bakersfield, CA	9,700	20.1	48,200
90	Lima, OH	9,500	48.2	19,700
91	Long Branch-Asbury Park, NJ	9,500	25.0	38,000
92	Terre Haute, IN	9,400	28.5	33,000
93	Spokane, WA	9,400	28.5	33,000
94	Beaumont-Port Arthur, TX	9,200	13.3	69,400
95	Honolulu, HI	9,200	13.3	69,400
96	Charlotte-Gastonia, NC	9,000	16.5	54,400
97	Evansville, IN-KY	8,700	34.3	25,400
98	Richmond, VA	8,700	18.6	46,800
99	Reading, PA	8,600	62.8	13,700
100	Knoxville, TN	8,500	24.4	34,900
101	Saginaw, MI	8,400	38.2	22,000
102	Kalamazoo-Portage, MI	8,400	34.0	24,700
103	Rockford, IL	8,300	31.4	26,400
104	Waterbury, CT	8,200	42.9	19,100
105	Johnson City-Kingsport-Bristol, TN-VA	8,200	23.1	35,500
106	Appleton-Oshkosh, WI	8,100	29.5	27,500
107	Fall River, MA-RI	8,000	58.8	13,600
108	Racine, WI	7,900	45.9	17,200
109	Lorain-Elyria, OH	7,900	32.1	24,600
110	Greenville-Spartanburg, SC	7,800	16.3	47,800
111	New Bedford, MA	7,700	57.5	13,400
112	Battle Creek, MI	7,600	45.8	16,600
113	Poughkeepsie, NY	7,600	36.7	20,700
114	Shreveport, LA	7,600	19.9	38,200
115	New London-Norwich, CT-RI	7,500	35.0	21,400
116	Des Moines, IA	7,500	26.6	27,200
117	Stockton, CA	7,500	21.4	35,000
118	Hamilton-Middletown, OH	7,300	31.1	23,500

(continued on following page)

TABLE V-20. (continued)

Rank	SMSA	Children 0.5-5 Years Number in pre-1950 housing	Percent of total in SMSA	Total Number in SMSA
119	Wheeling, WV-OH	7,200	51.1	14,100
120	Springfield, OH	7,100	49.3	14,400
121	Stamford, CT	7,100	47.0	15,100
122	Lowell, MA-NH	6,900	43.1	16,000
123	Springfield, IL	6,900	42.6	16,200
124	Mobile, AL	6,900	15.5	44,600
125	Portland, ME	6,800	48.2	14,100
126	Joplin, MO	6,500	59.6	10,900
127	Jackson, MI	6,400	48.5	13,200
128	Manchester, NH	6,400	47.8	13,400
129	Muskegon-Norton Shores-Muskegon Heights, MI	6,400	40.5	15,800
130	Charleston, WV	6,400	30.3	21,100
131	Charleston-North Charleston, SC	6,400	15.8	40,600
132	Anderson, IN	6,200	47.3	13,100
133	Yakima, WA	6,200	37.3	16,600
134	McAllen-Pharr-Edinburg, TX	6,200	17.5	35,500
135	Austin, TX	6,200	13.6	45,500
136	Steubenville-Weirton, OH-WV	6,100	42.4	14,400
137	Chattanooga, TN-GA	6,100	22.1	27,600
138	Santa Rosa, CA	6,000	22.8	26,300
139	Visalia-Tulare-Porterville, CA	6,000	20.4	29,400
140	Portsmouth-Dover-Rochester, NH-ME	5,900	52.5	11,300
141	Salem, OR	5,900	24.2	24,400
142	Williamsport, PA	5,700	58.2	9,800
143	Janesville-Beloit, WI	5,700	45.2	12,600
144	Harrisburg, PA	5,600	54.9	10,200
145	Kenosha, WI	5,600	43.8	10,400
146	Decatur, IL	5,600	47.1	11,900
147	Modesto, CA	5,600	22.3	25,100
148	Glens Falls, NY	5,500	58.5	9,400
149	Elkhart, IN	5,500	41.0	13,400
150	Salisbury-Concord, NC	5,500	37.2	14,800
151	Sheboygan, WI	5,400	61.4	8,800
152	Johnstown, PA	5,400	56.8	9,500
153	Lancaster, PA	5,400	53.5	10,100
154	Savannah, GA	5,400	22.6	24,000
155	Sharon, PA	5,300	47.3	11,200
156	Elmira, NY	5,300	40.8	13,000

(continued on following page)

TABLE V-20. (continued)

Rank	SMSA	Children 0.5-5 Years: Number in pre-1950 housing	Children 0.5-5 Years: Percent of total in SMSA	Children 0.5-5 Years: Total Number in SMSA
157	Salinas-Seaside-Monterey, CA	5,300	18.9	28,000
158	Raleigh-Durham, NC	5,300	18.9	28,100
159	Baton Rouge, LA	5,300	10.8	49,100
160	Vallejo-Fairfield-Napa, CA	5,200	16.8	30,900
161	Benton Harbor, MI	5,100	32.9	15,300
162	Provo-Orem, UT	5,100	13.4	38,100
163	Orlando, FL	5,100	9.3	54,600
164	Altoona, PA	5,000	44.6	11,200
165	Green Bay, WI	5,000	30.5	16,400
166	Ann Arbor, MI	5,000	21.8	22,900
167	Jackson, MS	5,000	16.3	30,700
168	Sioux City, IA-NB	4,900	43.8	11,800
169	Galveston-Texas City, TX	4,900	28.7	17,100
170	Santa Cruz, CA	4,800	32.4	14,800
171	Parkersburg-Marietta, WV-OH	4,800	30.6	15,700
172	Lincoln, NB	4,800	28.6	16,800
173	Madison, WI	4,800	20.6	23,300
174	Oxnard-Simi Valley-Ventura, CA	4,800	9.8	48,900
175	Waterloo-Cedar Falls, IA	4,700	34.8	13,500
176	Montgomery, AL	4,700	38.3	24,000
177	Brownsville-Harlingen-San Benito, TX	4,700	17.7	24,000
178	Waco, TX	4,600	30.1	15,300
179	St. Cloud, MN	4,600	26.9	17,100
180	Springfield, MO	4,600	26.1	17,600
181	Newark, OH	4,500	44.1	10,200
182	Vineland-Millville-Bridgeton, NJ	4,400	38.6	11,400
183	Brockton, MA	4,400	36.4	12,100
184	Cedar Rapids, IA	4,400	30.6	14,400
185	Santa Barbara-Santa Maria-Lompoc, CA	4,400	30.6	14,400
186	Bloomington-Normal, IL	4,300	45.7	9,400
187	Wausau, WI	4,300	40.6	10,600
188	Fort Smith, AR-OK	4,300	23.9	18,000
189	Cumberland, MD-WV	4,200	57.5	7,300
190	St. Joseph, MO	4,200	55.3	7,600
191	Muncie, IN	4,200	38.2	11,000
192	Albuquerque, NM	4,200	10.5	40,000
193	York, PA	4,100	59.4	6,900
194	Eau Claire, WI	4,100	38.7	10,600
195	Atlantic City, NJ	4,100	29.5	13,900

(continued on following page)

TABLE V-20. (continued)

Rank	SMSA	Children 0.5-5 Years Number in pre-1950 housing	 Percent of total in SMSA	 Total Number in SMSA
196	Lake Charles, LA	4,100	24.3	16,900
197	Eugene-Springfield, OR	4,000	17.6	22,700
198	Mansfield, OH	3,900	38.2	10,200
199	Amarillo, TX	3,900	21.7	18,000
200	Columbia, SC	3,900	11.6	33,500
201	Kokomo, IN	3,800	36.5	10,400
202	Norwalk, CT	3,700	50.0	7,400
203	Fargo-Moorhead, ND-MN	3,700	28.5	13,000
204	Alexandria, LA	3,700	21.0	17,600
205	New Britain, CT	3,600	42.4	8,500
206	Kankakee, IL	3,600	34.0	10,600
207	Wichita Falls, TX	3,400	30.4	11,200
208	Roanoke, VA	3,400	19.1	17,800
209	Lakeland-Winter Haven, FL	3,400	15.0	22,700
210	Augusta, GA-SC	3,300	11.0	30,000
211	Bellingham, WA	3,200	38.1	8,400
212	Abilene, TX	3,200	29.9	10,700
213	Pueblo, CO	3,200	28.1	11,400
214	Columbus, GA-AL	3,200	14.5	22,000
215	Macon, GA	3,200	12.6	25,400
216	Anniston, AL	3,100	30.4	10,200
217	Biloxi-Gulfport, MS	3,100	17.1	18,100
218	Pensacola, FL	3,100	12.5	24,800
219	Huntsville, AL	3,100	11.7	26,500
220	State College, PA	3,000	41.7	7,200
221	Danville, VA	3,000	28.3	10,600
222	Greeley, CO	3,000	23.8	12,600
223	Fort Lauderdale-Hollywood, FL	3,000	4.9	61,300
224	Danbury, CT	2,900	25.4	11,400
225	Lubbock, TX	2,900	14.1	20,600
226	Hagerstown, MD	2,800	37.8	7,400
227	Anderson, SC	2,800	26.9	10,400
228	Gadsden, AL	2,700	28.1	9,600
229	Bay City, MI	2,700	25.5	10,600
230	Billings, MT	2,700	25.0	10,800
231	Champaign-Urbana-Rantoul, IL	2,700	20.9	12,900
232	Colorado Springs, CO	2,700	9.2	29,500
233	Tucson, AZ	2,700	6.3	46,100
234	Tyler, TX	2,600	21.1	12,300
235	Bremerton, WA	2,600	18.8	13,800
236	Sioux Falls, SD	2,500	26.0	9,600
237	Boise City, ID	2,500	15.2	16,400
238	Killeen-Temple, TX	2,500	10.6	23,600
239	Nashua, NH	2,400	25.5	9,400

(continued on following page)

TABLE V-20. (continued)

Rank	SMSA	Children 0.5-5 Years Number in pre-1950 housing	Percent of total in SMSA	Total Number in SMSA
240	Texarkana, TX-Texarkana, AR	2,400	21.8	11,000
241	Asheville, NC	2,400	20.0	12,000
242	Longview-Marshall, TX	2,400	15.7	15,300
243	Fayetteville, NC	2,400	8.5	28,300
244	Lafayette, LA	2,300	14.0	16,400
245	Lafayette-West Lafayette, IN	2,200	24.4	9,000
246	Lynchburg, VA	2,200	20.8	10,600
247	Wilmington, NC	2,200	17.3	12,700
248	Daytona Beach, FL	2,200	13.8	15,900
249	West Palm Beach-Boca Raton, FL	2,200	6.5	35,600
250	Medford, OR	2,100	16.4	12,800
251	Fayetteville-Springdale, AR	2,100	13.5	15,500
252	Richland-Kennewick-Pasco, WA	2,100	12.7	16,500
253	Burlington, VT	2,000	20.8	9,600
254	Yuba City, CA	2,000	18.5	10,800
255	Florence, AL	2,000	16.9	11,800
256	Rock Hill, SC	1,900	24.7	7,700
257	Hickory, NC	1,900	18.6	10,200
258	Monroe, LA	1,900	14.6	13,400
259	Chico, CA	1,800	17.3	10,400
260	Bradenton, FL	1,700	17.9	9,500
261	Petersburg-Colonial Heights-Hopewell, VA	1,700	15.7	10,800
262	Athens, GA	1,700	15.6	10,900
263	Newport News-Hampton, VA	1,700	9.8	17,400
264	Las Vegas, NV	1,700	4.3	40,000
265	Tallahassee, FL	1,600	11.0	14,500
266	Melbourne-Titusville-Cocoa, FL	1,600	9.2	17,300
267	Albany, GA	1,500	12.7	11,800
268	Tuscaloosa, AL	1,500	11.4	13,000
269	Reno, NV	1,500	11.3	13,300
270	Ocala, FL	1,400	16.9	8,300
271	Olympia, WA	1,400	13.0	10,800
272	Anchorage, AK	1,400	7.0	20,100
273	Redding, CA	1,300	12.4	10,500
274	Gainesville, FL	1,300	11.1	11,700
275	Fort Collins, CO	1,300	9.5	13,700
276	Clarksville-Hopkinsville, TN-KY	1,300	9.1	14,300
277	Odessa, TX	1,200	9.9	12,100
278	Florence, SC	1,000	9.7	10,300
279	Jacksonville, NC	1,000	8.5	11,800

(continued on following page)

TABLE V-20. (continued)

		Children 0.5-5 Years		
Rank	SMSA	Number in pre-1950 housing	Percent of total in SMSA	Total Number in SMSA
280	Charlottesville, VA	900	13.8	6,500
281	Fort Myers-Cape Coral, FL	800	5.7	14,100
282	Columbia, MO	600	6.7	8,900
283	Sarasota, FL	400	4.3	9,300
284	Pascagoula-Moss Point, MS	400	3.1	12,900
	COMBINED SMSAs			
285	El Paso, TX & Las Cruces, NM	10,300	15.4	66,800
286	Greensboro-Winston-Salem-High Point & Burlington, NC	7,400	19.7	37,500
287	Fitchburg-Leominster & Pittsfield, MA	6,400	49.6	12,900
288	Bangor & Lewiston-Auburn, ME	6,300	54.3	11,600
289	Bristol & Meriden, CT	5,400	46.2	11,700
290	Bismarck, ND & Grand Forks, MN	5,100	25.6	19,900
291	Dubuque & Iowa City, IA	5,000	36.0	13,900
292	Lawton & Enid, OK	4,600	25.1	18,300
293	Laredo & Victoria, TX	4,000	18.6	21,500
294	La Crosse, WI & Rochester, MN	3,900	25.3	15,400
295	Little Rock & Pine Bluff, AR	3,700	25.2	14,700
296	Bloomington, IN & Owensboro, KY	3,700	23.6	15,700
297	Casper, WY & Great Falls, MT	3,500	20.5	17,100
298	Midland & San Angelo, TX	2,900	18.2	15,900
299	Topeka & Lawrence, KS	2,800	20.3	13,800
300	Bryan-College Station & Sherman-Denison, TX	2,400	17.1	14,000
301	Ft. Walton Beach & Panama City, FL	2,000	9.6	20,800
	TOTAL	4,374,600	30.6	14,278,900

Thirty-three SMSAs and one paired SMSA showed 50% or more of all children living in pre-1950 housing units, with Jersey City, NJ, showing 72.6%. Among the 122 SMSAs for which we could separate "Inside Central City/Outside Central City" status, we found 14 SMSAs where 70% or more of the inner-city children lived in pre-1950 housing. In Buffalo, NY, 84.8% of inner-city children lived in the oldest housing.

TABLE V-21. CINCINNATI, OHIO-KENTUCKY - SMSA 1980 CENSUS COUNT OF CHILDREN OF ALL RACES 0.5-5 YEARS OLD BY FAMILY INCOME, URBAN STATUS, AND AGE OF HOUSING

	Number of Children				Percent			
Strata	Pre-1950	1950-1969	1970-1980	Total	Pre-1950	1950-1969	1970-1980	Total
Inside Central City								
<$6,000	4,200	4,300	800	9,300	21.8	35.2	38.1	27.7
$6,000-14,999	4,800	3,400	300	8,500	24.9	27.9	14.3	25.3
≥$15,000	10,300	4,500	1,000	15,800	53.4	36.9	47.6	47.0
Total	19,300	12,200	2,100	33,600	100.0	100.0	100.0	100.0
Outside Central City								
<$6,000	2,200	2,900	1,900	7,000	8.8	8.7	5.7	7.7
$6,000-14,999	8,000	4,900	4,700	17,600	32.0	14.8	14.2	19.3
≥$15,000	14,800	25,400	26,600	66,800	59.2	76.5	80.1	73.1
Total	25,000	33,200	33,200	91,400	100.0	100.0	100.0	100.0
Total SMSA								
<$6,000	6,400	7,200	2,700	16,300	14.4	15.9	7.6	13.0
$6,000-14,999	12,800	8,300	5,000	26,100	28.9	18.3	14.2	20.9
≥$15,000	25,100	29,900	27,600	82,600	56.7	65.9	78.2	66.1
Total	44,300	45,400	35,300	125,000	100.0	100.0	100.0	100.0

Tables showing the relationship of family income and housing age for the children showed, first of all and not surprisingly, that children in the highest income group constitute the majority of the children in the SMSAs. Consequently, these children frequently are a large portion of residents in each of the three ages of housing categories (Table V-21, complete sets in Appendices A, B, and C). Exceptions are SMSAs of more recent growth where such children tend to live in the suburban areas and are found relatively infrequently in recently constructed housing in the central city. In the other SMSAs, children tend to be distributed more in relation to their proportions in the child population. Table V-21 is a detailed illustrative compilation for housing age and family income in the SMSA for Cincinnati, OH-KY.

The tables in Appendices A, B, and C show the distribution of the three income levels in the column "Percent-Total" and the rows labeled "Total SMSA" for the larger SMSAs and the single set of rows for the smaller SMSAs. We found that while the children from the highest income population lived disproportionately more frequently in the newest and newer housing, they still represent a significant portion of the residents in the oldest housing as well.

D. SUMMARY AND OVERVIEW

Discussion of Sections A, B, and C and a summary are presented here. Given the diverse topics, each section is first discussed separately.

1. Lead-Exposed Children in SMSAs

Every effort was made to establish accurate numbers for the population bases used to project the numbers of children exposed to lead levels described in Chapter V, Section A.

The number of children in each SMSA constituting the base populations, for which the strata were isolated and to which the prevalences were applied, consist of actual U.S. Census counts and counts based on legal documents, i.e., birth and infant death certificates. In these enumerations, the only sources of variation for our purposes were the rounding off to the nearest hundred and applying population strata distributions four years earlier than 1984. These two factors would have minimal impact on the accuracy of compiling the base population. The time discrepancy results in a conservative outcome since an economic recession occurred in the intervening years, and poverty is associated with higher prevalence of higher Pb-B levels.

Applying prevalences at indicated Pb-B criterion values involved using the relevant original NHANES II data base in tandem with a projection process to establish prevalences for 1984. The projections use the original data set, which consists of actual measurements obtained during 1976-1980, the field phase of the survey. Projections were made via logistic regression analysis and were employed to update prevalences for Pb-B levels of 15, 20, and 25 µg/dl. The reasons for selecting these Pb-B values are discussed in Chapter IV and the problem of over- and underestimates was discussed in the first section of Chapter V.

Projection of the NHANES II prevalences to 1984 is the principal phase of the process that includes the element of uncertainty always associated with any estimation process. Given the universally recognized high quality of the design and execution of the NHANES II survey and the relatively good reliability of logistic regression analyses, the results presented in Section A of this chapter are reasonable.

We employed U.S. Census data base populations within strata and the projected Pb-B prevalences to estimate the number of children with Pb-B above selected levels at various levels of geographic differentiation. Early efforts were made to estimate the number of exposed children in each of the 318 SMSAs. Such an approach subsequently was judged to contain too much uncertainty and was abandoned. Next, an attempt was made to derive projected prevalences for each of the four major U.S. regions identified and employed in the NHANES II survey, and to combine the rates with the stratified SMSA populations in each of the four regions. Generating satisfactory projections at the regional level was not possible for several reasons. We therefore calculated the numbers of white and black children as a function of Pb-B levels and the nationwide total population of the SMSAs. These stratified exposure estimates appear in Tables V-4, V-5, V-6, V-7, and V-8.

The estimates presented in Tables V-4 through V-8 show that criterion Pb-B levels representing health risks are found in all child population segments, not only in the target populations usually defined by the screening programs as being at risk. These target populations constitute a large component of the population at risk, but they do not, by any means, constitute that entire population.

The large projected numbers of children at risk among those with suburban and other "Not Inside Central City" residences are shown in Table V-5 and V-8. The prevalences for these children are lower than those for "Inside Central City" children, but this is countered by the finding that more children live in the suburbs in most, though not all, SMSAs. Hence, the estimates reflect both of these key factors.

Table V-8 shows the overall dimensions of the problem among the SMSA child population, as well as the distribution by "Inside Central City" and other residence when available. About 13,840,000 young black and white children lived in all SMSAs in 1984, and 2,381,000, or about 17% were estimated to have Pb-B levels above 15 µg/d, reasonable estimates for white and black children living in SMSAs in 1984. Furthermore, when balancing the specific factors leading to overestimations and underestimations discussed before, these estimates for white and black children in SMSAs should be considered as the best estimates that can be made based on available scientific data.

2. Numbers of Lead-Exposed Children Detected by Screening Programs

Over the years, lead-exposure screening efforts and the factors related to them have varied considerably. These factors include defining the risk of toxicity, administrative organization, and amount of funding.

When CDC administered the screening programs, there were more than 60 active programs with an established and fairly rigid reporting protocol. At present, there are about 40-45; but the exact number is difficult to identify since only states report and reporting is voluntary. Programs now are supported by state-based block grants controlled by state agencies, and only states submit screening results rather than each program as was done under CDC administration. The state agencies should be commended for these efforts, but the numbers being reported may be less representative of the exposure problem than were obtained under central CDC control and with uniform criteria. These changes have made it difficult to estimate time trends, determine prevalences, and compare current prevalences with those obtained under prior, CDC-administered programs.

For the most recent level of screening activity, the results of ATSDR's December 1986 survey of existing programs show that for 1985, 785,285 children were screened in 40 or more programs and that the overall rate of elevated exposure cases was 1.5%. In terms of numbers, more children appear to be screened at present, although statistical comparisons with the older CDC framework would be difficult.

Toxicity responses for 1985-1986 ranged from 0.3% for four programs to 11.0% for the City of St. Louis program. The five highest prevalences are 11.0% (St. Louis), 9.0% (Augusta and Savannah, GA), 4.9% (Harrisburg, PA), 3.5% (Washington, DC) and 3.5% (Merrimac Valley, MA, a program within a MCH project). Most of the rates for confirmed toxicity cases were below 2%.

Prevalences reported for earlier years include those from state agencies, as summarized by the Public Health Foundation. For FY 1983, reporting agencies screened 675,571 children and recorded a prevalence of 1.5%. Similarly, the CDC data for FY 1981 indicated that 535,730 children were screened, with 21,897 meeting the toxicity risk criteria then in use--a prevalence of about 4%. For the reasons noted above, we cannot closely compare these three sets of results.

Trends in prevalences over time, at least in the years under CDC control, suggest a moderate decline in toxicity risk from 1973 on. Data from two programs for 1973 to 1985, and data from the St. Louis, MO, program show downward shifts, even with changes in risk classifications.

With the 1985 change in the CDC toxicity risk classifications, we might expect the number of toxicity risk positive results to increase, and the New York City screening program results show such an increase. In future years, then, the prevalences will reflect this change in classification, but they will also reflect the impact of reduced levels of lead in gasoline and food.

3. Comparison of Prevalences Found in NHANES II Updated Prevalences and U.S. Screening Programs

Because the NHANES II prevalences, and therefore the updated adjusted rates, are based on Pb-B determinations for the subjects, there is consequently no intervening problem of first determining EPs. These rates are not subject to systematic underreporting due to false negative results found when EP determinations are used to classify the initial population group being evaluated and identify who will then be tested for Pb-B levels. Screening programs, however, test for EP first.

Other factors in screening programs probably produce lower prevalences compared with those that can be predicted on the basis of results from NHANES II and other surveys. Screenings are not always intensive, for example, house-to-house; in most cases, they are conducted in clinics. Any factor that affects the characteristics of clinic visits also affects Pb-B prevalences. For example, the mothers who bring children to clinics may be more concerned, informed, and motivated than those who do not bring their children.

Finally, communities with the highest prevalences may not have unique situations. Their approach to the problem simply may be more systematic than in other communities, and therefore, they report the highest rates. The more appropriate question may well be why all communities do not have higher rates rather than why certain communities have high rates. This type of question cannot be answered unless the screening programs are again centrally administered.

4. Children With Potential Exposure to Leaded Paint in the SMSAs

This section summarizes an attempt to identify potential lead exposure by each SMSA and by a specific source, leaded paint. It therefore combines approaches dealing with source-specific exposure described earlier in Chapter V and those in Chapter VI.

The number of children living in pre-1950 housing was obtained from the actual 1980 Census enumeration. Table V-20 and Appendix D present the ranking of SMSAs by the number of children in pre-1950 housing and show that young children are least often found in the newest housing, built in 1970-1980. This is probably due to the fact that young families are least able to afford newer housing and particularly newer units inside central cities. While children in families with the lowest incomes were found disproportionately in the older housing, large proportions of children in the highest income families were also found living in the oldest housing stock. Furthermore, the children with family incomes above the poverty levels frequently constituted the largest proportion in the oldest housing.

While there is no perfect correlation between the age of the housing and the presence of a leaded paint hazard, note that weathering and chalking occur even when there is no dilapidation or deterioration of the housing. We do not know how many pre-1950 housing units have been renovated or had high lead-content paint removed, but we can say with some confidence that the fraction is quite low, given data for Massachusetts cities described in Chapter IX.

Equally, we do not know the consequences of such paint removals in terms of increasing the raw lead content of the sites' dusts and soils within reach of these young children.

Another factor to consider when examining the significance of housing age is the presence of lead in plumbing and the potential contamination of the drinking water. Older housing may contain lead plumbing, and the most recent stock may contain lead in the solder used for copper piping. In either case, lead may leach into the drinking water, which is discussed more fully in Chapter VI.

The environment in which young children are housed, which depends on the income of young families at the start of the family phase of the life cycle as well as the availability of housing units of different ages, exposes a large proportion of young children regardless of family income to the older housing stock that in turn is likely to contain the paint with the highest lead content. The ubiquity of Pb-B levels representing health risks found by the NHANES II survey is supported by the finding that young children frequently live in the type of housing most likely to contain sources of lead: in paint, drinking water, and dust/soil.

Finally, one significant but often unrecognized point about childhood exposure sources such as leaded paint deserves emphasis. Until leaded paint and contaminated dust and soil are removed from young children's environment, the number of young children at risk for adverse health effects will accumulate over time and continue to present a serious public health problem since each new cohort of children will in turn be exposed to lead in the environment. In other words, the cumulative tally of exposed children becomes much larger as time passes than the specific counts given at one point in time. This is due to both the high mobility within the high leaded paint zones in which the high-risk families live and the sociological fact that poor families may be "locked" into such housing for many years.

5. Conclusions and Overview

This report represents the first systematic effort to quantify the extent of the U.S. child lead-poisoning problem and to place such numbers in some context of distribution of the children, the lead sources, the adverse health responses, and strategies for lead reduction or removal. This chapter is a key component of this effort and is important both for the numbers provided and for helping answer the obvious questions "Which children have the problem?" and "What can we start to do about it?"

From the key findings of Chapter V, we conclude that the total number of U.S. children exposed to lead at unacceptable levels (Pb-B >15 µg/dl), 2.4 million SMSA children or 17% of the SMSA child total, arise from many different socioeconomic and demographic strata.

It was to be expected that the "traditional" high-risk groups, e.g., poor, inner-city black children, would have figured prominently in the estimation outcomes, and they do. These high-risk groups are usually defined as such in terms of high prevalence rates of elevated Pb-B levels. Less well understood, perhaps, is the fact that the totals for exposed strata in this chapter are derived from both a prevalence for a given Pb-B and the base population by which the prevalence fraction is multiplied to give a stratum final total.

The consequences of an estimating exercise, across strata, for exposure totals is simply that large numbers in a stratum's base population can have quite low prevalences for certain Pb-B levels and still yield numbers that are comparable to those obtained from high-risk strata that have smaller base populations of children but quite high prevalences of elevated Pb-B levels.

In Chapter V, a variety of estimating strategies were employed, and provide quite different numbers for exposure estimates. These differences were explained earlier in the summary. Furthermore, some of the totals complement each other, providing different views of the same total population of U.S. children. For example, examining the very detailed U.S. Census Bureau counts (not estimates) of children in the 318 SMSAs reported in terms of housing age and family income (Section C) produces the unexpected finding that more children in older housing (high paint-lead levels) were also in noncentral-city, nonpoverty families than were children associated with the typical risk groups. This observation corresponds to this report's projected Pb-B distributions in the nation's children.

These distributions in high-risk housing might account for why certain Pb-B prevalences in the otherwise lower-risk strata of U.S. SMSA children are as high as they are. In other words, distribution of the nation's children into high lead-exposure risk housing is uniform enough that all strata of such children, when examined by means of a national composite survey such as NHANES II, will produce significant prevalences. Clearly, however, other sources also have an impact on Pb-B levels and their prevalences in the general child population, and this point is established in Chapter VI.

At present, the third national survey of its type, NHANES III, is in the planning stage, and eventually results of this survey (expected in the mid-1990s) will produce more precise numbers for prevalences of Pb-B levels in strata used in NHANES II and in this report. In the interim, the 1990 U.S. Census will also be conducted. For the present, however, the estimates and U.S. Census counts of children in Chapter V are the best that can be done with the available data.

VI. EXAMINATION OF NUMBERS OF LEAD-EXPOSED U.S. CHILDREN BY LEAD SOURCE

A. GENERAL ISSUES

Three general points need to be discussed before source-specific childhood lead exposure can be addressed. First, "exposure" must be defined according to the available data and the intent of the Congressional directive. Second, a relationship must be established between the lead source and the exposure population to assess the biological diversity of human population response. Third, behavioral characteristics and other covariates influencing the degree to which exposure to lead in the external environment results in internal (systemic) exposure must be studied.

1. The Level of Exposure Risk in Human Populations Characterized and Quantified by Lead Source

Source-specific exposures are difficult to delineate because of multimedia exposures to lead. When exposures come from several sources, how should they be ranked? For example, children exposed to lead in paint (by either direct chewing or swallowing paint chips) often simultaneously contact dust from chalked or weathered paint. Therefore, one source of lead may be the dominant but not the sole source. For rural or suburban adults, food or water can be a major source of lead. It is difficult to quantify exposure once the important sources have been determined. Section 118(f) places no statutory restraints on defining exposure and estimating the number of children exposed. As noted in Chapter II, human exposure can be indexed either by external or internal means--that is, environmental or biological monitoring.

General environmental monitoring can estimate external exposure in the population. However, these estimates are very broad because they estimate the number of subjects at the lead source, regardless of the level of contact with the source. Although these estimates produce the largest numbers, they are the least accurate when associating exposure with the actual risk of toxicity. For example, when leaded paint exposure is estimated in this way, houses containing lead paint are counted and the number of children from the U.S. Census count is distributed proportionally among them.

A more accurate exposure assessment can be achieved if individuals are exposed only to a source containing lead levels exceeding that needed to elevate Pb-B levels. This estimate is determined by some empirical relationship, usually through use of regression equations. Lead levels in all old leaded paint are high enough for this estimate; so are lead levels in some dust and soil contaminated from lead fallout in air or weathering paint (U.S. EPA, 1986a). Blood lead from water and food can also be assessed using this method.

The most accurate way to assess exposure for source-specific lead is to study elevations in a biological indicator. For example, determining if elevated Pb-B levels can be traced to the intake or uptake of source-specific lead.

Reliable information is needed when using biological monitoring to estimate lead-exposed populations by source. This reliable information requires some means of apportioning a given Pb-B level among input sources. When available, this information would be invaluable in reporting the true scope of source-based lead poisoning in the United States. In view of this, we need to discuss the relationship of Pb-B level to sources in more detail.

The levels of precision in assessing population exposures by lead source are given in Table VI-1. This table summarizes the type of exposure analysis needed for each level of precision within a given source category. From the table, one can determine the approximate exposure level associated with each type of estimation. Those children estimated to be exposed internally (*in vivo*, systemically) at unacceptable levels of Pb-B are of greatest concern. Those children estimated to have a measurable increase in systemic exposure, i.e., elevated Pb-B levels, but who have not been characterized by risk level for adverse effects achieved by the increase are of second greatest concern. Finally, those children estimated to be in an external lead environment and who risk internal contact with the toxicant, although no data indicating systemic uptake exist, are the third concern. Children with potential exposure are at higher risk than children in environments with no lead.

This hierarchical array of types of exposure assessment is illustrated in Table VI-1 for lead in drinking water. In Section F of this chapter, estimates are given for: (1) number of children in an environment likely to have potential exposure to elevated levels of lead in water; (2) number of children who will have some degree of actual exposure due to elevated lead levels in water, but exposures not necessarily high enough to produce lead poisoning. Finally, (3) number of children who have varying elevated lead exposures (Pb-B elevations) to lead levels in water above the proposed maximum contaminant level (MCL) of 20 μg/dl.

2. Relationships of External to Internal Lead Exposure on a Total Population Basis

Before applying any biological indicator for lead (such as Pb-B) to population surveys or to categorize risk, its quantitative dimensions--especially the distribution of Pb-B levels among that population--must be understood. Populations show a range of responses, including a range of Pb-B levels, rather than a single uniform response to lead exposure. This variability of responses occurs because individuals within a population react in different ways to the same exposure, because of host factors. Also, the inherent nature of toxicant distribution, as affected by toxic cellular or organ responses, can internally increase toxicant levels enough to affect their distribution, which is called dose-dependent distribution.

In humans, Pb-B values are distributed in a log-normal rather than a normal manner. A log-normal distribution, unlike a normal distribution, is skewed and not symmetrically bell-shaped. One consequence of this skewing, which is crucial to public health, is that the segment with highest blood lead levels, the upper tail of this distribution, includes a larger fraction of the entire population than it would in a normal distribution. The actual fraction of these individuals in a given population can be defined by the geometric standard deviation (GSD) and the geometric mean (GM). Risk assessors or risk managers consider this segment when defining some desirable cut-off value in a cumulative frequency to protect a risk population at some selected exposure level, such as some specified Pb-B level.

From the GSD, percentages of populations at risk can be calculated as well as the mean or median population lead burden, which is required for the

TABLE VI-1. CATEGORIES OF ESTIMATION METHODS FOR CHILDREN EXPOSED TO LEAD BY SOURCE

Source Category	Level of Precision	Method of Exposure Measurement
1. Lead in paint	Potential exposure	Determination of numbers of children in housing with highest likely lead-paint burdens; complements Chapter V data
	Potential exposure with a better indication of actual exposure risk	Number of children estimated to be in deteriorated housing with leaded paint: peeling paint, broken plaster, other damage
	Likely actual exposure	Use of a specifically determined prevalence for an NHANES II stratum matching such children; other, regional survey data
2. Lead in gasoline	Potential exposure (Pb-B changes) in a subset of U.S. urban child population	Total number of young children in the 100 largest U.S. cities
	Actual exposures based on leaded gasoline combustion	Logistic regression analysis to estimate numbers of children below selected Pb-B criterion values
3. Lead from stationary sources	Potential exposure	Total of young children in communities near lead operations
	Actual exposure	Prevalence of indicated Pb-B levels, at or above some criterion level in actual field studies of stationary sources
4. Lead in dusts and soils	Potential exposure	Summation of potential exposure numbers from the above three categories

(continued on following page)

TABLE VI-1. (continued)

Source Category	Level of Precision	Method of Exposure Measurement
	Actual exposure	Summation of corresponding actual exposure numbers from first three actual exposure categories, or use of multimedia regression equations (not possible with present data)
5. Lead in drinking water	Potential exposure	Numbers of young children in homes with either old lead plumbing or with lead solder in new home
	Actual exposure that is measurable but not highest toxicity risk	Numbers of young children in homes with lead levels in drinking water above 20 μg/l
	Actual exposure at or near toxic levels	Number of children estimated from NHANES II prevalences of projected toxic Pb-B levels
6. Lead in food	Potential exposure at or near toxic levels	Number of U.S. children within selected age group
	Actual exposure	Fraction of those potentially exposed children whose food lead intake may raise Pb-B high enough to cause concern

next step in establishing regulations. The maximum amount of lead that regulators permit in a lead source without exceeding these mean or median values can also be computed. These regulated levels usually correspond to the amount of anthropogenic activity, such as emissions from industry and auto exhaust. If, by using these calculations, it is determined that, for example, 99% of the U.S. population lies below a specific Pb-B ceiling level, about 2.4 million individuals would still be considered to have an unacceptable level of toxicity risk.

These observations are relevant for lead sources that are ubiquitous in the United States, but which show "moderate" concentrations when given as the average Pb-B resulting from a specific source. The propensity to assess the extent of the lead problem with these averages can be misleading. Because lower averages apparently would suggest a lower public health risk, they can result in misleading interpretations when studying inherently serious effects or when there are tens to hundreds of millions in the population being discussed. The reasons for why this is so follow from the discussion below.

Figure VI-1 illustrates a hypothetical blood lead distribution for a human population. The quantitative usefulness of such a figure can be seen in the 1986 Draft Report of the World Health Organization for air quality guideline recommendations in the European community (WHO, 1986). In developing guidelines for lead levels in air for Europe, the WHO Working Group for lead took several approaches; one approach determined the following parameters.

(1) Based on available evidence at the time (September 1984), the group determined that a Pb-B level of 20 µg/dl was the exposure level of action (WHO, 1986).

(2) The group also determined that this Pb-B 20 µg/dl value was to be prevented in 98% of the European community populations.

(3) Because only data on adults were available, the requisite GM and GSD data were employed to determine that a 98% cumulative frequency for a 20 µg/dl Pb-B level required a median Pb-B value of 10.5 µg/dl in adult populations. In other words, for the Pb-B level in 98% of an adult population to remain below 20 µg/dl, that population requires a median Pb-B level of 10.5 µg/dl.

These three parameters are depicted in Figure VI-1. The shaded area represents subjects in the remaining 2% of the distribution above the level of 20 µg/dl.

If we were to apply this approach to the U.S. population, we would first determine--using the lowest of the three projection formulae of the U.S. Census Bureau--that the 1985 projected adult population is 175 million. If we also allow 98% to have levels below 20 µg/dl, with a 10.5 µg/dl median, the remaining 2% still gives 3.5 million individuals at some risk of lead toxicity. If we expand the protection (cumulative frequency) band to 99.5% and/or reduce the acceptable Pb-B level to 15 µg/dl or less, then this median will be considerably below 10.5 µg/dl.

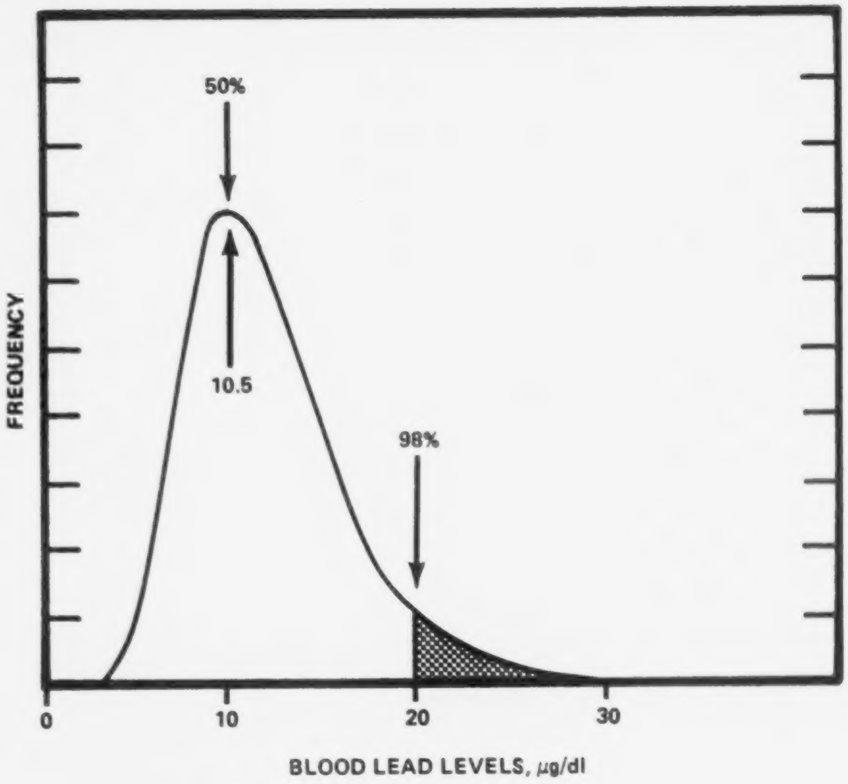


Figure VI-1. Illustrative log-normal Pb-B distribution curve. Values from WHO (1986): Median = 10.5 µg/dl., 98% = 20.0 µg/dl. Shaded area = 2%.

How do these estimates fit available information on the recent or current median Pb-B values for adults in the U.S. population? In the WHO/UNEP world survey of Pb-B levels, included in the Global Environmental Monitoring Survey (GEMS), the U.S. adult median Pb-B level in 1981 was 7.5 µg/dl (Friberg and Vahter, 1983). Judging from the various projections and other data from the U.S. EPA (1985, 1986a), this median Pb-B value has declined in recent years.

For a given Pb-B level and a given cumulative frequency, the values for children would probably be higher than for an adult population, because distributions of Pb-B levels among members of a population are also age-dependent; that is, young children have a different distribution from adults, especially in heterogeneous populations. Adult/child differences occur even in rather homogeneous populations. U.S. EPA (1986a) shows these differences using NHANES II data (Table VI-2).

TABLE VI-2. SUMMARY OF Pb-B LEVELS (μg/dl) OF A RELATIVELY HOMOGENEOUS WHITE POPULATION IN THE UNITED STATES[a,b]

Age Group (yr)	Geometric Mean (μg/dl)	Median (μg/dl)	99th Percentile (μg/dl)	Geometric Standard Deviation (μg/dl)
0.5-6	12.9	13.0	32.0	1.43
6-18	10.6	10.0	24.0	1.46
18+ (Men)	14.7	15.0	35.8	1.44
18+ (Women)	10.0	10.0	23.0	1.46

[a] Adapted from U.S. EPA (1986a). EPA internal analysis of available NHANES II data sets.

[b] Sample size (vertical order): 752, 573, 922, and 927.

From a toxicological standpoint, the distribution of lead levels within target organs (the central nervous system of children, for example), as a function of a given Pb-B value is important for illustrating the distribution phenomena. These distributions are not found in present epidemiological approaches to lead exposure. However, their importance can be seen by studying another metal toxicant, cadmium. Kjellstrom (1985) discusses target organ distributions of cadmium (measured in vivo) including biological indicators and population dose-response curves.

The distribution of lead in major body components such as bone is another important biological factor, because lead can become mobilized and re-enter the blood (Chapters III and IV). The characteristics of this distribution in young children over time are of particular interest.

3. Human Behavior and Other Factors in Source-Specific Population Exposures to Lead

In the relevant literature, discussed in detail by U.S. EPA (1986a) and in critical studies and reviews cited in the EPA document, we find that, given a specified and significant degree of external lead contamination, a number of socioeconomic/demographic variables can affect the relationship between lead in the environment and blood in children. The interrelationships of several of these variables can amplify the degree of adverse interaction between the child and the contaminated environment. Such factors affect the relative results that investigators find in relating such exposure indices as Pb-B to some measure of adverse effect. For example, one can understand that if children are residing in a heavily contaminated environment and are at the age when they are orally exploring their environment, then the degree of lead exposure via such exploration will be influenced by parental attention to child activity, extent of mouthing, and ingestion of lead-containing material. We might then expect inverse relationships between quality of parental care and degree of lead exposure and some measure of outcome, at least under conditions of moderate lead exposure. Such studies, however, do not imply that lead exposure does not occur or does not significantly contribute to an adverse effect. They simply imply that the degree of exposure interacts with other factors.

Examining modifying factors for scientific reasons is appropriate and necessary, but we should not assume that it will neutralize the effect of low-level exposure in the overall lead problem. These assumptions would be illogical and can detract from a simple rule of health risk management: abating the lead sources removes or reduces the risk for all children, whatever their socioeconomic or demographic status. Such caveats against misinterpretations of the above-noted studies are even stronger when we examine the significant rise in the number of lead toxicity cases associated with urban "gentrification," where children of upper socioeconomic status families reside in lead-contaminated environments formerly occupied by children of lower socioeconomic status (Rabinowitz et al., 1985).

4. Organization of the Chapter

In the main body of this section we estimate and discuss the numbers of children exposed to six different lead sources: paint, gasoline combustion, stationary emissions, soil or dust, water and food. We do not include relatively limited sources of lead (such as exposure from painted toys or hobbies) or contact specific to an ethnic group (as seen in some types of folk medicine). This approach does not imply that these sources are unimportant in certain circumstances, particularly with newly arrived ethnic groups. However, these sources are difficult to quantify and do not affect the overall effect of the major sources of lead described below. For some of these six categories--lead in food, for example--we cannot identify any specific inputs; we can only say that human activity, collectively, adds considerably to lead levels. The relative impact of these lead sources varies greatly, both by source and by different geographic/demographic/socioeconomic strata. These strata refer to numbers of subjects and not necessarily to the intensity of exposure at a contaminated site. Any population of children having significant contact with lead in dust and soil is also highly likely to have significant contact with lead in air and paint. This category, however, is mainly included to identify a significant pathway for childhood lead toxicity and to evaluate the source for dust and soil in linkage with its primary sources.

B. NUMBERS OF CHILDREN EXPOSED TO LEAD IN PAINT

Many reports address the role of leaded paint in lead poisoning and consider paint lead poisoning to be a public health issue (see, for example, CDC, 1985; U.S. EPA, 1986a). The cause-and-effect relationship of leaded paint to severe lead poisoning dates back many decades. Evidence has long been available to show radio-opaque (lead) paint chips in the abdomens of children who had both high Pb-B levels and severe poisoning and who had not been in contact with any other source of lead. Although the total number of acute, very severe U.S. cases of lead poisoning has declined greatly, the basic epidemiological picture characterizing paint lead-associated toxicity has not materially changed for chronic interaction. The problem can still be described as it has been in some recent studies.

In their prospective study of inner-city children in Cincinnati, OH, Clark et al. (1985) found that child Pb-B levels varied across housing categories and children who lived in the worst housing had the highest Pb-B levels. The housing-quality category accounted for more than 50% of the Pb-B variability in 18-month-old children. In a prospective examination of Baltimore children treated for lead toxicity, Chisolm et al. (1985) observed that children returned to housing where work had been done to remove leaded paint showed significantly higher Pb-B levels than children returned to public housing free of leaded paint. Furthermore, little decline was noted in the Pb-B levels of children who lived in lead-abated units over an extended period--indicating that current efforts to abate lead fall short of public health goals. Both lead paint-contaminated and deteriorated housing units are included in the lead problem. These units have the largest exposures and they also represent affordable housing for a sizable fraction of inner-city children and parents.

Public health officials have long viewed leaded paint as a lead source in the child's home. However, they should also consider older public buildings used as day-care centers, kindergartens, elementary schools, etc., as potentially serious exposure hazards.

1. Estimation Strategies and Methods

As indicated in Table VI-1, estimates of U.S. children exposed to lead in paint are based on degrees of potential risk and on estimates of the numbers of children predicted to have actual elevated risk because of paint-associated elevations in their Pb-B levels. For potential risk of lead exposure via leaded paint, estimates are given for children living in units with leaded paint and children living in units with an elevated probability of actual exposure because of peeling paint, broken plaster, or other deterioration. The data sets used include calculations by Pope (1986) and the estimates of categories and numbers of lead-painted units with problems from the American Housing Survey of the U.S. Bureau of the Census, 1983 (U.S. Bureau of the Census, 1986).

Pope (1986) first determined a child density factor for each unit (i.e., numbers of children per lead-painted residence) by examining the child population under 7 years of age and the number of housing units in the nation. The child-density factor is specifically the ratio of children under 7 years of age per 1000 housing units. This number, given by Pope (1986), is 287/1000 or 0.287. National figures for housing yielded a value for the fraction of housing units containing leaded paint as a function of age: pre-1940, 1940-1959, and 1960-1974. Furthermore, data from the American Housing Survey, U.S. Bureau of the Census, provide three criteria for unsound units that are relevant for lead paint exposure: peeling paint, broken or cracked plaster, and holes in walls. The data source also provides the fractions of the total units that these units represent. We therefore have estimates of: (1) the total number of children in homes with lead paint; and (2) the number of children in homes with leaded paint that are in disrepair, thus maximizing lead exposure. In addition to Pope's best estimate, we also used Pope's national upper bound. Pope also estimated children by four major regions: Northeast, Midwest, West, and South.

There is a general dearth of nationwide studies that estimate the number of children living in lead paint-containing homes who have elevated Pb-B levels, and much of the information relating leaded paint in the environment to Pb-B levels is not in a form suitable for our analyses. Reasonable data for our needs are available in two forms. The first is a comprehensive unit-by-unit screening conducted in Chicago in 1978 as part of the city's lead-screening program for that year. Although screening was confined to one metropolitan area, it was a comprehensive study, involving more than 80,000 housing units, to determine both Pb-B levels and the presence of leaded paint in the children's houses. The second approach projected (to 1984) prevalences from NHANES II data for Pb-B levels in those socioeconomic/demographic strata where paint is likely to be the major, if not entire, source of exposure. These prevalences were presented in the previous chapter.

EPA's Office of Policy Analysis (U.S. EPA, 1985) used the Chicago data to estimate the likely percentage of children in Chicago (under 6 years old) who would have a Pb-B level greater than 30 µg/dl due to leaded paint exposure. Using estimates of the probability of lead in paint occurring in a home with a child having lead toxicity and the probability of lead in paint occurring in the survey housing in general--both parameters were determined in the Chicago survey--EPA employed Bayes' theorem to determine the probability of elevated Pb-B at the then current toxicity risk level. This prevalence value, 12.8% of all children in the survey, has limited use since it is a dated estimate, represents a Pb-B level too high for our present purposes, and may not represent a best estimate for that year.

One can, alternatively, use prevalences for more appropriate Pb-B levels than the rather high Chicago survey criterion value of 30 µg/dl. In Chapter V, prevalences updated to 1984 are tabulated at Pb-B levels of >15, >20, and >25 µg/dl for young children in various socioeconomic/demographic strata. We have taken the number of young children enumerated by the U.S. Census as living in deteriorated housing with 100% high lead paint, and applied the most logical prevalences for the stratum that would apply to children in deteriorated housing. We assumed that children in 100% deteriorated, high lead-paint housing conform to the stratum that is in the inner city, in the densest population areas, and in the lowest income category. We also assumed that many of these children would be black.

From the relevant tabulation in Chapter V, the Pb-B prevalences (Pb-B level percentage) for the 0.5 to 5-year-old/inner-city/higher urban density/lowest income/black stratum are: >15 μg/dl, 67.8%; >20 μg/dl, 30.8%; >25 μg/dl, 10.6%.

2. Results

To estimate the total numbers of young children living in lead-paint housing, we can first estimate the percentages of housing having paint with lead greater than or equal to 0.7 mg/cm^2 as: pre-1940, 99%; 1940-1959, 70%; and 1959-1974, 20% (Pope, 1986). Given a total housing inventory of 80,390,000 in 1980 (U.S. Bureau of the Census, 1983 Survey), we arrive at a final tally of 41,964,000, or 52% of all residential housing units have lead paint greater than or equal to 0.7 mg/cm^2. This figure for the lead-paint concentration is based on the 1985 CDC statement (CDC, 1985). A count of children less than 7 years old in homes with lead paint is shown in Table VI-3, as given by Pope (1986). The national best estimate of the number of children in all lead-painted housing, regardless of the age or state of repair, is 12,043,000. The national upper-bound estimate is higher--13,579,000. When compared to the number of children ranked by age of housing and SMSA as given in Chapter V, and taking into account some differences in children's age in these two sets

TABLE VI-3. NATIONAL BEST ESTIMATE AND UPPER BOUND OF NUMBERS OF CHILDREN UNDER 7 YEARS OLD IN LEAD-BASED PAINTED U.S. HOUSING BY AGE OF UNITS[a]

Estimate Type	Housing Age	Number of Lead-based Painted Houses (Thousands)	Number of Children (Thousands)
Best Estimate			
	Pre-1940	20,505	5,885
	1940-1959	16,141	4,632
	1960-1974	5,318	1,526
Total	Pre-1980	41,964	12,043
Upper Bound			
	Pre-1940	20,712	5,944
	1940-1959	20,753	5,956
	1960-1974	5,850	1,679
Total	Pre-1980	47,315	13,579

[a] Adapted from Pope (1986).

of data, we then can obtain the percentage of all young children who reside in urbanized housing. This percentage is about 80%.

In considering the numbers of children who live in deteriorated housing that contain leaded paint, Pope (1986) classified the housing according to Census Bureau designations for unsound housing within the three house age groups. Table VI-4 shows the best national estimate and the national upper bound for children in deteriorated, lead-painted houses and the number of these houses as a function of age and condition, along with the totals. From Table VI-4, we can calculate that the best national estimate and the national upper bound estimate of children under 7 years old living in unsound lead-painted housing are 1,772,000 and 1,996,000, respectively. Table VI-5 shows young children exposed to peeling paint (the only sign of deterioration) in lead-based painted homes by the four major geographic regions, as given by Pope (1986). As expected, the older developed areas, specifically urban areas in the Northeast and Midwest, have the highest and next highest figures: Northeast, 174,000; Midwest, 139,000 for children in homes with peeling leaded-paint as the survey criterion for deterioration. The South with 130,000 ranks third, while the West has the lowest figure, 77,000.

TABLE VI-4. NUMBERS OF U.S. CHILDREN RESIDING IN UNSOUND AND LEAD-BASED PAINTED HOUSING RANKED BY AGE AND CRITERIA FOR DETERIORATION[a,b,c]

Unsound Category	Age of Home	Number of Unsound Lead-Based Painted Houses	Number of Children
Peeling paint	Pre-1940	964,000	277,000
	1940-1959	758,000	218,000
	1960-1974	250,000	72,000
Total	Pre-1980	1,972,000	567,000
Broken plaster	Pre-1980	1,594,000	458,000
Holes in walls	Pre-1980	2,602,000	747,000
Grand Totals	Pre-1980	6,199,000 (6,954,000)[d]	1,772,000 (1,996,000)[d]

[a] Adapted from Pope (1986).

[b] Housing data from 1983 Housing Survey, (U.S. Bureau of the Census, 1986).

[c] Children under 7 years old.

[d] National upper bound to the numbers.

TABLE VI-5. REGIONAL BEST ESTIMATE OF NUMBERS OF CHILDREN IN UNSOUND, LEAD-BASED PAINTED HOUSING BY AGE AND NUMBERS OF PEELING PAINT UNITS[a,b;c,d]

Region	Age	Number Peeling Paint Lead-Based Painted Houses	Number of Children
Northeast	Pre-1940	432,000	110,000
	1940-1959	203,000	51,000
	1960-1974	51,000	13,000
Total	Pre-1980	686,000	174,000
Midwest	Pre-1940	264,000	74,000
	1940-1959	159,000	47,000
	1960-1974	47,000	14,000
Total	Pre-1980	479,000	139,000
West	Pre-1940	92,000	27,000
	1940-1959	127,000	37,000
	1960-1974	44,000	13,000
Total	Pre-1980	236,000	77,000
South	Pre-1940	156,000	46,000
	1940-1959	203,000	60,000
	1960-1974	80,000	24,000
Total	Pre-1980	439,000	130,000

[a] Adapted from Pope (1986).

[b] Estimates of housing from U.S. Census Bureau (1983).

[c] Children under 7 years of age.

[d] These figures do not include children in units meeting other criteria for unsoundness but only for peeling paint.

One can ask whether these figures are not actually lower bounds when compared with estimates that might be made by summing, across SMSAs, all young children in the known socioeconomic/demographic risk categories. Sums of the SMSA-specific tabulation in Chapter V, Section C, indicate that this is not the case. The estimates of housing (based on Pope) used here include non-SMSA housing stock. Non-SMAS housing should not be in any better condition than SMSA housing. For example, the fraction of substandard homes in rural America is about 41% (2+ million/5 million), according to Lerman (Economic Research Service, USDA, 1986). "Substandard" is technically different from "unsound" in these surveys, but the fraction of houses with peeling paint and cracked plaster as well as lead-based painted surfaces is probably significant in substandard rural housing. Toxicologically, these factors determine the level of lead exposure, not definitions of housing, per se. We may be placing a lower bound to the child density per unit by simultaneously increasing the number of units, thereby offsetting distortions. The above estimates of numbers of children potentially exposed to leaded paint minimize the contributions to the total numbers that would arise from children exposed to lead because older housing is being renovated--the so-called urban gentrification phenomenon. Reliable figures for quantifying this aspect of childhood lead exposure are not available.

In considering estimates of these children exposed to lead in paint who have elevated Pb-B levels because of this exposure, we first chose to combine the numbers in Tables VI-4 and VI-5 for children in unsound, lead-based painted housing with the 12.8% of children >30 μg/dl Pb-B calculated by EPA for all children residing in these units who represent a large urban area. This approach gives an estimate of about 230,000 children; however, the relative accuracy is unknown for reasons already stated. Next, the results of using selected lower Pb-B criterion values--15, 20, and 25 μg/dl--using NHANES II projected prevalences for values that would be plausible for a group of children living in 100% deteriorated, high lead-based painted housing combined with base numbers of such children are tabulated in Table VI-6. The numbers for Table VI-6 are reasonable estimates but are still likely underestimates (see next paragraph). The rationale for assuming the demographic/socioeconomic profile of children likely to reside in such housing is also reasonable. The number of children in such housing having Pb-Bs above 15 μg/dl is around 1,200,000 while the corresponding figures for Pb-B limits of above 20 and 25 μg/dl are around 545,000 and 188,000, respectively.

Numbers in Table VI-6 do not give us an estimate of exposed children in old housing with high paint lead levels but lacking specific criteria for deterioration. The totals in this case may be substantial, since Section C, Chapter V noted that many families in old housing are not in the central city and not in poverty and the homes of these children are not in a deteriorating state. For this reason, these figures in Table VI-6 should be viewed as possible lower bounds (or underestimates) to the true count. Similarly, the stratum of NHANES II selected as appropriate for assignment of these children in order to obtain actual prevalences may represent percentages, after projection to 1984, which are actually from a mix of housing quality. In other words, the true projected Pb-B prevalences for present-day children in 100% deteriorated, high lead paint housing may be considerably above that set of Pb-B prevalences actually selected for estimates in Table VI-6.

TABLE VI-6. ESTIMATED NUMBERS OF U.S. CHILDREN LIVING IN UNSOUND, LEAD-BASED PAINTED HOUSING ABOVE INDICATED Pb-B CRITERION VALUES[a,b]

Category	Housing Age	Total Children	Children With Pb-B (μg/dl)		
			>15	>20	>25
Peeling paint	Pre-1940	277,000	187,800	85,200	29,400
	1940-1959	218,000	147,800	67,100	23,100
	1960-1974	72,000	48,800	22,200	7,600
Total	Pre-1980	567,000	384,400	174,500	60,100
Broken plaster	Pre-1980	458,000	310,500	140,900	48,500
Hole in wall	Pre-1980	747,000	506,500	229,800	79,500
Grand Total		1,772,000	1,201,400	545,200	188,100

[a] Total child count from Table VI-4.

[b] Selection of NHANES II stratum for use of specific prevalences is discussed in text. Prevalences are from Table V-1.

Using both of these factors, the true count of children with elevated Pb-B levels could be underestimated considerably. On the other hand the estimates may overlap in Table VI-6. Units with peeling paint may also have been counted as having broken plaster, etc., in a number of instances.

C. NUMBERS OF CHILDREN EXPOSED TO LEAD FROM LEADED GASOLINE

The combustion of leaded gasoline by motor vehicles and the dispersal of lead from exhausts have had a major role in the status of lead as a public health issue. The recent debate on air quality criteria, health effects, and the leaded gasoline phasedown is only the latest episode of a controversy dating back to 1925, when lead additives had just been introduced. The Hamilton et al. (1925) review of the new leaded gasoline problem from a public health standpoint voiced many concerns that applied equally to the 1950s, 1960s, or 1970s (for a public health perspective on 60 years of leaded gasoline, see Rosner and Markowitz, 1985).

Since about 60 years ago when lead additives for gasoline were introduced, millions of tons of lead from the combustion and dissipation of leaded gasoline in the United States have entered the environment. Much of this quantity has been lodged in ecosystems where it can lead to human exposure, for example, through dust and soil. A chronological look at such inputs is provided in Table VI-7. For the 10 years shown in Table VI-7, more than 1 million metric tons of gasoline lead were dispersed just in the United States.

A comparison of leaded paint and leaded gasoline indicates the full, insidious nature of the lead problem. Leaded paint can cause very high exposure, with overt poisoning, in a rather confined area and can also induce chronic toxicity due to lower persistent exposure. In contrast, lead from vehicular exhausts can cause sufficient exposure, related to chronic health effects, over a large area.

Evidence showing that leaded gasoline was enough of a human health risk to require further regulatory changes in the existing ambient air standard was compiled in the 1977 EPA Air Quality Criteria for Lead (U.S. EPA, 1977). In addition to assessing the scientific literature related to leaded gasoline, the document also included concepts and perceptions that were then beginning to figure in health risk assessment and biomedical practice as applied to environmental health. Relevant environmental health phenomena, such as blood lead distribution in the population, were discussed. Aggregate exposure to pollutants was defined and discussed. In addition, subtle adverse effects of lead--particularly as noted by the pediatric medical community--were examined.

TABLE VI-7. RECENT CONSUMPTION OF LEAD IN GASOLINE[a]

Calendar Year	Leaded Gasoline Volume (10^9 gal)	Lead Consumed (10^3 ton)[b]
1975	92.5	167.4
1976	87.0	171.4
1977	79.7	168.9
1978	75.0	153.0
1979	68.1	129.4
1980	57.5	78.8
1981	51.0	60.7
1982	52.5	59.9
1983	47.5	52.3
1984	43.8	46.0
Total	654.6	1,087.8

[a] From U.S. EPA (1986a).

[b] Consumption in metric tons.

Between the 1977 and 1986 EPA Air Quality Criteria for Lead Documents, information on the lead problem accumulated; much of it concerned lead from the combustion of leaded gasoline. For example, the quantitative relationships of blood lead to this source were studied; the knowledge about the adverse effects of lead at lower levels was expanded; and the mechanisms of toxicological action were examined. Most of these subjects lie beyond the scope of this report.

Gasoline lead makes a sizable contribution (about 90 to 95%) to the total atmospheric lead burden in developed countries such as the United States. By using lead isotope ratio tagging for lead in gasoline, we can follow that fraction of lead not only into the environment but also into humans. The best estimate of leaded gasoline contributions, using isotope ratios in urban Italy, is about 90% (Fachetti and Geiss, 1982).

As expected, air lead levels related to gasoline combustion and auto density are highest in areas of highest traffic volume, urban and suburban commuting, and commercial activity zones. The most extensive data set of U.S. ambient air levels over the years was compiled by the National Filter Analysis Network and its predecessors. Such surveys have shown that ambient air-lead levels in remote parts of the United States are 2 ng/m^3 and that in urban areas, levels are often 1 to 3 μg/m^3, some 1,000-fold higher. The trend in these air-lead levels is downward, particularly with the leaded gasoline phasedown that EPA implemented in about 1975. The current allowable lead content of leaded gasoline is 0.1 g/gallon (F.R., 1985, March 7). From 1975 to 1984 U.S. gasoline lead consumption decreased 73%, and estimated lead levels in ambient air showed a similar decrease.

Dispersal of gasoline-based lead from air into food, soils, and dust via fallout has been amply documented and critically evaluated (U.S. EPA, 1986a; WHO, 1986). Airborne lead fallout associated with traffic, as well as lead levels in exterior dust, house dust, soil, and plants are highest near traffic arteries in urban and suburban areas. This observation parallels the findings for ambient air lead from urban stationary sources, such as secondary smelters and municipal incinerators.

The quantitative relationships between airborne lead from leaded gasoline combustion or other sources and a biological indicator such as Pb-B have been the object of numerous studies, and they are discussed at some length in Chapter 11 of U.S. EPA (1986a). This report focuses on the relative impact of airborne lead inhaled directly and air lead ingested after fallout, such as by children who take in dust and soil.

Given the continuing interest in the impact of airborne lead on Pb-B levels, numerous studies have been conducted on the blood lead to airborne lead ratio; that is, the amount of change in Pb-B one might expect from a unit change (μg/m^3) in airborne lead. Basically, such a ratio is an oversimplified, only partially integrative depiction of systemic exposure. It permits a quick, but imprecise, look at the effects of changes in a community's airborne lead levels on the systemic exposure of child and adult populations.

The blood lead/airborne lead ratio is lowest when (1) the ratios are examined experimentally in test chambers where the only exposure pathway is inhaled airborne lead, or (2) the ratios are examined for adults, a group with little secondary entry of airborne lead via dust and soil. This ratio rises considerably when children are comprehensively examined for all impacts of airborne lead (Brunekreef, 1984). Direct inhalation of airborne lead yields ratios of about 1 to 2. When children are examined for both direct (inhalation due to emitted lead particulates) and indirect (entrained dust/soil inhalation or ingestion of dust and soil) effects, a ratio of 5 to 6 or even higher is measured. That is, for each 1 μg/m^3 increase, the Pb-B level rises 5 to 6 μg/dl. Because dust ingestion and gut absorption vary so much among children, the contribution to individual children varies widely around these figures.

Since internal, or systemic, lead exposure comes from several sources, it is necessary to determine how much exposure comes exclusively from airborne lead. This measurement was conducted using an isotope tracing method, the isotope lead experiment (ILE, Fachetti and Geiss, 1982). Such studies indicate that airborne lead contributes at least 20 to 25% of total Pb-B in adults with inhalation as a principal route. Such estimates are considered lower bounds since a significant fraction of lead within this isotopic ratio, when absorbed, moves to the bone, where it joins a large lead burden. This mixture now loses any isotope identity in blood by a significant "isotope dilution." A sizable fraction of this short-term lead deposit will move from the bone back into blood (U.S. EPA, 1986a).

Collectively, the above data indicate (1) that past gasoline lead inputs produced airborne lead that added significantly to atmospheric and soil/dust/ food burdens; (2) airborne lead added significantly to blood lead by direct and indirect routes, yielding 20 to 25%, as a lower bound, based on isotope tracing and up to 50% based on NHANES II data. In children, blood lead/airborne lead ratios of 5 to 6 and even higher indicate that the airborne lead input to blood lead can be very significant. Therefore, we would expect alterations in airborne lead, paralleling reductions of lead in gasoline, to reduce lead in blood. This relationship is indeed the case, and major support for this statement is the very high correlation between the decrease in Pb-B levels in the general population seen in the NHANES II survey for all segments of the population and declines in the use of leaded gasoline. Specifically, the correlation indicates that decreased use of leaded gasoline over 1976-1980 is the reason for the lower Pb-B levels. Regional data supporting the above national trend were presented by Rabinowitz and Needleman (1982) for a large sampling of newborn cord blood levels in the Boston, MA, area.

1. Estimation Strategies and Methods

If we examine the total potential of direct (inhalation) and indirect (fallout) childhood exposure to leaded gasoline, we mainly examine large urban-suburban areas with denser traffic, i.e., urban population centers, in which airborne lead levels have been high enough to add a potentially significant burden to dust and soil. Since such parts of the environment can retain lead for long periods (U.S. EPA, 1986a), a population of children can be exposed to lead lingering from this source long after airborne lead levels have started to decline.

To examine the number of children 6 years old or younger who were potentially exposed to airborne lead via inhalation or dust/soil lead, children less than 7 years old in the 100 largest U.S. cities were counted from Census Bureau 1984 estimates of total population, and 11% of the population was determined to be under 7 years old.

No children are exposed to lead exclusively from leaded gasoline, and we estimate imprecisely the fraction of children exposed to lead via this source. We can conclude, however, that children whose blood lead levels have changed due to the decreased use of leaded gasoline can be said to have sufficient contact with lead by this route to meet the intent of Section 118(f). As noted earlier, Pb-B levels are declining, and efforts have been directed to quantitate this decline as a function of the decreased use of leaded gasoline. As part of this effort, EPA's Office of Policy Analysis (U.S. EPA, 1985) performed projection analyses of the number of children whose Pb-B levels will fall below selected criterion values due to continued declines in leaded gasoline use over a number of years.

The EPA Office used logistic regression analyses based on NHANES II data. These regressions were estimated for both black and white children and for the rather broad age band of children at risk, 6 months to 13 years old. Assuming that a log-normal Pb-B distribution would occur with the decreased use of leaded gasoline, EPA generated estimates of the mean and variance of transformed (normal) distribution for determining percentages above specific Pb-B levels, logistic regression estimates of the children with Pb-B equal to or above 30 μg/dl, and computer estimates of the mean of the log-normal distribution (SAS/SURREGR). A more detailed discussion of this method is beyond the scope or purpose of this report, and appears in the original EPA document (1985). Since much of this methodology is relevant to Chapter V, these techniques also appear in Appendix G of this report.

2. Results

As indicated, we restricted our count of children at risk of exposure to leaded gasoline to the 100 largest U.S. cities. These cities had a 1984 estimated total population of 50,597,300, of which 5,565,700, or 11%, were children less than 7 years old. This estimate of some 5.6 million children at risk cannot be related directly to the following discussion on the impact of leaded gasoline on Pb-B levels, because the children are different ages and possess inherent differences.

Table VI-8 shows the numbers of U.S. children, 6 months to 13 years old, whose Pb-B levels will fall below selected toxicity levels when projected to 1990. The numbers are significant, showing expected increases as the Pb-B level is lowered. Smaller numbers of children occur at higher Pb-B criterion levels, since the numbers originally above these levels were smaller. These values represent nationwide projections for a 13-year age band in children. Since Pb-B distributions are age-dependent in this age band, especially in younger children, it is not easy to divide out these figures into narrower age bands from the values in Table VI-8. One difficulty with the broad age range for the number of affected children is that the numbers of very young children are not available to the reader. A second factor, evident in Table VI-8, is the prevalence of the numbers of children with Pb-B levels below expected Pb-B levels in the years long after the the leaded gasoline phasedown: gasoline

TABLE VI-8. ESTIMATED NUMBERS OF U.S. CHILDREN (THOUSANDS) FALLING BELOW INDICATED Pb-B (μg/dl) LEVELS AS A RESULT OF Pb-GASOLINE PHASEOUT[a,b]

Blood lead (μg/dl)	1985	1986	1987	1988	1989	1990
25	72	172	157	144	130	119
20	232	563	518	476	434	400
15	696	1,726	1,597	1,476	1,353	1,252

[a] From U.S. EPA (1985). Based on regulatory action beginning January 1, 1986, to achieve 0.1 g/gal by January 1, 1988.

[b] Tabulations in original U.S. EPA (1985) analysis were extended only to 1990 for this table.

lead phasedown alone will not bring all Pb-B levels down to acceptable low levels. Table VI-8 shows, even in terms of the 25 μg/dl level, that sizable numbers of children are projected to fall below these various Pb-B ceiling levels from 1986-1990 and that large declines are expected for criterion levels of 15 and 20 μg/dl.

D. NUMBERS OF CHILDREN EXPOSED TO LEAD FROM STATIONARY EMISSION SOURCES

As noted in Chapter II, stationary sources mainly refer to fixed operations that emit lead into the atmosphere and, consequently, into other ecological areas. Such sources include primary and secondary smelters, incinerators, and operations involved in coal and waste oil combustion. In terms of impact, these operations mainly affect neighboring communities, but they can cause severe lead contamination.

The United States has 11 mines, 5 primary smelters and refineries, 60 secondary smelters, and 132 plants where lead-acid batteries are manufactured. For these sources, we have to consider that contamination occurs even after the facilities have closed. Over the years, a lead-emitting operation will add a heavy ecological burden to nearby areas. Of particular concern is lead fallout from smelters transferring to nearby soil, dust, and forest cover. The evidence linking lead emissions from stationary operations to the elevated body lead burdens of young children is well established. These connections have been derived from studies of a number of major U.S. smelter operations (including ones in Idaho, Montana, and Nebraska) that have been extensively examined by U.S. EPA (1986a). Airborne lead levels near lead smelters and refineries, and in some cases up to 5 to 10 km away, reached 5 to 15 µg/m³ in past periods, particularly before emission controls were installed in the 1970s. In impact zones, lead levels ranged up to 100,000 ppm (10% by weight) where emission controls were minimal, and near smelters in Missouri, soil lead levels reached levels up to 60,000 ppm. Today, soil and dust levels range between 500 and 5,000 ppm in areas near point sources. The levels decrease exponentially with distance from the operation.

Results of numerous studies document that children sustain marked increases in blood lead and body lead burdens when they live near stationary lead emitters, particularly lead smelters. This relationship can be seen in the investigations of Yankel et al. (1977), who evaluated Pb-B levels in children 1 to 9 years old living near a smelting operation in Silver Valley, ID, in 1974-75. Table VI-9 shows the results of the biological and environmental monitoring in this smelter area. These overall blood lead results were extremely high, especially the percentage of levels over 40 µg/dl. Airborne lead levels were also very high, even at 10 km from the operation. In the zone adjacent to the smelter, about 100% of the Pb-B levels were above 40 µg/dl. Elevated Pb-B levels have been found in children living near other smelter sites, both in the United States and elsewhere (U.S. EPA, 1986a).

TABLE VI-9. GEOMETRIC MEAN Pb-B LEVELS (µg/dl) BY DISTANCE FROM SMELTER (AREAS 1-6) FOR CHILDREN[a] NEAR IDAHO SMELTER[b]

Area and km from Source		Airborne Lead (µg/m³)	Geometric Mean Pb-B (GSD) (µg/dl)	% Pb-B >40
1	0-1.6	18.0	65.9 (1.30)	98.9
2	1.6-4.0	14.0	47.7 (1.32)	72.6
3	4.0-10.0	6.7	33.8 (1.25)	21.4
4	10.0-24.0	3.1	32.2 (1.29)	17.8
5	24.0-32.0	1.5	27.5 (1.30)	8.8
6	~75	1.2	21.2 (1.29)	1.1

[a] Ages 1-9 years.

[b] EPA analysis of Yankel et al. (1977) data.

Recent surveys carried out in two smelter communities, Montana (CDC, 1986a) and Idaho (CDC, 1986b), as a joint effort by CDC, EPA, and the respective states, indicate that considerable levels of residual dust and soil contamination linger after former active, high atmospheric inputs.

As a consequence of this lingering exposure problem in a smelter community in Idaho (CDC, 1986b), the survey found that:

(1) Children who lived close to the smelter in the Silver Valley area of Idaho had a higher geometric mean Pb-B (20 μg/dl) than children farther away (11 μg/dl).

(2) Detailed statistical analysis of the data base showed that the only significant environmental contributor was lead in the soil, and its contribution was via lead in household dust. Soil lead near the smelter had a geometric mean level of 3,472 ppm, while a mean level of 481 ppm was measured for sites farther away; the corresponding geometric mean for lead dust was 3,933 near the smelter and 1,138 ppm farther away.

(3) At the time of the survey, the ambient air level had mean values, by area, of 0.10 to 0.28 $\mu g/m^3$.

(4) The percentage of children living near the smelter whose Pb-B and EP levels exceeded the CDC risk criteria for 1985, i.e., Pb-B ≧25 μg/dl and EP ≧35 μg/dl, was 26%, while the corresponding figure for those farther away was 2%.

These two comprehensive studies conclusively document that previous lead fallout remains a main contributor to lead exposure in general, and contributes to body lead burden of children in particular.

We turn now to estimates of U.S. children who are either potentially at risk to lead exposure from fixed operations or actually have elevated Pb-B levels due to exposure from stationary sources. Such population data are surprisingly meager. Only a limited number of reports have helped us to quantify this aspect of the U.S. lead problem. One preliminary report, for EPA's Office of Air Quality Planning and Standards (OAQPS) (GCA Corporation, 1986), provides the larger of the two data sets for the total number of children living near stationary sources in potential exposure to lead. The data set of the Lead Industries Association (TRC Environmental Consultants, Inc., 1986) provides a markedly different estimate of children from that of EPA. These data sets are for potential exposure.

1. Estimation Strategies and Methods

The interim OAQPS numbers are derived from dividing the stationary sources into three categories; primary smelters, secondary smelters, and lead-acid battery plants. Estimates for each group were collected for different radii around the operations, which reflect differences in lead dispersal patterns. The numbers are for total subjects and the fraction of children less than 7 years old, 10.4%.

The LIA study differs from the OAQPS study in terms of inventory of units still operating, quadrants surveyed, and radii around the operations. The LIA study mainly made its estimates based on ambient airborne lead levels. The LIA assessment did not count closed facilities. But in assessing the net and continuing lead exposure of children around stationary operations, closed facilities must be included because past lead emissions continue to have an impact. These sources must be included to avoid underestimating the risk of lead exposure.

The LIA study narrowed the radius of exposure population considerably, compared with the OAQPS model. The LIA considered airborne lead movement for the dominant wind direction at the emission point, but did not allow for changes in wind direction on soil and dust levels in sectors not in the dominant path.

Data in Table VI-9 show that at 10 to 24 km away from a smelter, with an airborne lead level of 3.1 $\mu g/m^3$, the geometric mean Pb-B level in children was 32.2 µg/dl, with almost a fifth of the lead levels above 40 µg/dl. If children are also examined for uptake pathways 10 to 24 km from the smelter--distances well beyond both the LIA and the OAQPS plottings--the Pb-B/Pb-Air ratio is almost 6 when the levels are normalized to those of a group of control children in this zone. This ratio strongly suggests heavy additional input into Pb-B above that contributed by inhalation, i.e., dust and soil lead contributions. Angle et al. (1984), for an Omaha smelter area, reported a value of 6 to 7 for total inputs to children's Pb-B levels by lead exposure through air inhalation, dust, and soil.

The above estimates by LIA and OAQPS were for potential exposure subjects. Actual Pb-B prevalence data for such sites as primary and secondary smelters also exist. The recent smelter community studies in Montana and Idaho (CDC, 1986a, 1986b), as cited earlier, show that both dust and soil levels and Pb-B levels of children remain elevated even when airborne lead levels have dropped to very low levels (0.10 to 0.28 μg/m³). These results showed that about 1% (1/98) of the children within 1 mile (1.6 km) of the East Helena smelter and 26% (11/43) of the children within 1 mile (1.6 km) of the Kellogg smelter met the 1985 CDC criteria for some level of lead toxicity: a Pb-B level of at least 25 μg/dl and an EP level of at least 35 μg/dl. Further, results of a systematic survey in Dallas, TX showed 4% of the children living near secondary smelters in the area had Pb-B levels above 20 μg/dl (City of Dallas, 1985).

2. Results

Table VI-10 shows the values from the LIA study (TRC, Inc., 1986) for potential numbers of exposed children; Table VI-11 shows the interim OAQPS estimates of the same potential exposure group (GCA, 1985). The OAQPS data are probably more useful in accounting for the indirect impact of stationary source emissions on present and past levels in dust and soil. If results of other studies, such as the Yankel et al. (1977) Silver Valley, ID, study and the CDC East Helena, MT, and Silver Valley, Idaho studies (CDC, 1986a, 1986b), are examined together, the OAQPS tabulation may be a conservative estimate of the total exposure population for primary smelters. Dispersion radii for secondary smelters are usually shorter than for primary smelters, but such operations tend to be in densely populated urban areas. OAQPS is reanalyzing and updating information on impact zones around different point sources.

Table VI-11 provides the best guide for estimating the total number of children who may have potential lead exposure due to proximity to stationary sources. To estimate actual exposures, we used the rates of 1% and 26% for primary smelters and 4% for secondary smelters, as noted earlier. These rates give differences among the point source emission characteristics, geography, neighboring communities, etc. Extrapolating the results of the East Helena primary smelter study--where 1% of the children had Pb-B levels over 25 μg/dl--yields a total of 210 children for all primary smelters. The corresponding prevalence recently found in Idaho, 26%, gives a figure of around 5,500. In 1984, in Herculaneum, MO, 18% of the children living within 1.5 miles of the primary lead smelter had Pb-B levels above 25 μg/dl (communication, OAQPS/EPA to ATSDR). Using this factor yields a total of around 3,800. Likewise, an estimate of 7,500 is the corresponding figure for children sufficiently affected by secondary smelters to have Pb-B levels above 20 μg/dl,

TABLE VI-10. EXTRAPOLATED PEDIATRIC POPULATION ESTIMATES FOR STATIONARY LEAD SOURCES: TRC/LIA AND EPA APPROACHES[a,b]

Source	Operating Facilities	Number of Children Extrapolated for TRC Distances[c]		Extrapolation for EPA Distances[c]
		1.0 $\mu g/m^3$	0.5 $\mu g/m^3$	
Primary smelter	5	616	1,946	6,154
Primary refinery	1	7	553	8,961
Secondary smelter	23	141	2,377	19,738
Tetraethyl lead	1	37	122	365
Battery Plants	98	1,647	3,293	15,827
Total		2,448	8,291	51,045

[a] Adapted from TRC, In: LIA study as submitted to OAQPS/EPA (TRC, 1986).

[b] Using 8,291 as the best estimate of potentially exposed subjects.

[c] Distances from point source as defined in EPA and TRC reports.

TABLE VI-11. GCA/OAQPS ESTIMATES OF TOTAL AND CHILD (≤7 YEARS) POPULATIONS EXPOSED TO STATIONARY SOURCES OF LEAD[a]

Source	Radius Around Plant (km)	Total Population	Number of Children
Primary lead smelters	5	200,000	21,000
Secondary lead smelters	2	1,800,000	187,000
Lead-acid battery plants	1	240,000	25,000
Total		2,240,000	233,000

[a] As tabulated and submitted to OAQPS/EPA, April 8, 1985. Radii to estimate potentially affected population are preliminary and are under re-examination.

assuming a 4% prevalence above 20 µg/dl reported for the Dallas survey. We cannot estimate the numbers with elevated Pb-B levels near the lead-acid battery plants because data do not exist.

E. NUMBERS OF CHILDREN EXPOSED TO LEAD IN DUSTS AND SOILS

In previous sections, the three major contributors to lead in dusts and soils were evaluated according to a ranking of childhood lead exposure by source: lead paint, gasoline lead, and stationary lead emissions. Dusts can be further classified into soil dusts, street dusts, and household dusts. In various exposure environments, the relative importance of these types for childhood exposure differs. In summer, street dusts are probably more important because of the amount of time children spend outdoors; in colder weather, household dusts are probably more important. Children may be further exposed to lead in dust via dust brought home on the clothing of working parents and relatives. These occupational dusts may have high lead content, reflecting fairly concentrated amounts of substances such as lead oxide (Milar and Mushak, 1982). As noted earlier, lead levels in household and street dusts vary as a function of their primary contributors; these levels can range well above 1,000 ppm in many urban areas (see U.S. EPA, 1986a, for a detailed discussion). Brunekreef et al. (1983) determined, in a study in the Netherlands at sites without major point sources, that household dust increased in lead content by 400 to 700 ppm for each 1 µg/m^3 increase in airborne lead.

A major problem with reported studies of lead in dust and soil in relation to body lead burdens is the absence of any current standard method for collecting samples at the test site. For soils, current core samples or surface scrapings can be taken; multiple sites can be sampled or hot spots can be emphasized.

Near stationary lead sources, lead levels in dust respond more dramatically to changes in airborne lead levels (Yankel et al., 1977; CDC, 1986a, 1986b). Soil lead content is also considerably affected by airborne lead fallout from mobile and stationary sources. Lead levels in soil can rise considerably with fallout, but the levels generally are lower than in dusts because the nonlead fraction dilutes the soil samples more (see, e.g., CDC, 1986b). An important aspect of soil contamination is the uptake of lead onto plants, which is mainly deposited on the surface with some uptake through the root system. Plant uptake becomes significant when assessing lead exposure in plants that are an important part of the human diet. Lead in soil also impacts lead levels in livestock; when the animals forage lead-contaminated crops or ingest soil lead, lead may enter the food chain.

A number of reports have addressed the quantitative relationships of lead in soil and dust to Pb-B, and U.S. EPA (1986a) has summarized these reports. In general, lead in dust and soil at levels of 500 to 1,000 ppm begins to affect children's Pb-B levels (Baker et al., 1977; Mielke et al., 1984); a number of investigators have found a highly significant correlation between Pb-B levels and lead levels in dust and soil (see, for example, Angle et al., 1984; Roels et al., 1980).

Results from U.S.-based investigations of these relationships have been confirmed and extended to other countries (Duggan and Inskip, 1985).

In the study of the Silver Valley, ID smelter reported by CDC (1986b), the difference in the Pb-B means for children near the smelter operation vs. those farther away was 9 μg/dl (20 μg/dl vs. 11 μg/dl, respectively). The average soil levels for the sites differed as much as 3,000 ppm. Calculations show that a 1 μg/dl rise occurred in the Pb-B level for each 330 ppm of soil; the corresponding relationship for lead in dust was essentially the same, 310 ppm.

Recent data of Bornschein et al. (1987b) and Clark et al. (1987) show that lead in soil and paint contributes to lead in dust; dust lead transmitted via children's hands to their mouths accounts for a significant fraction of Pb-B increases. Also, an increase of lead by 1,000 ppm raises Pb-B by 6.2 μg/dl.

The relationships of lead in soil and dust to Pb-B levels in various studies (see U.S. EPA, 1986a; Bornschein et al., 1987b) show a range of values, generally changing between 3 and 7 μg/dl for every 1,000 ppm change. Note that some of these studies derived Pb-B response data for Pb-B levels which are higher than those now judged as unacceptable. In the Baker et al. (1977) study, for example, lead levels in dust above 1,000 ppm caused a rise above 40 μg/dl. This observation implies that a lower Pb-B threshold for reference would have been associated with lead levels below 1,000 ppm.

A determination of the direct and indirect contributions of airborne lead to Pb-B, that is, the fraction from direct inhalation and that from fallout, has been noted in the lead isotope ratio study in Turin, Italy (Fachetti, 1985); results showed that 60% of the amount enters adult subjects via inhalation and 40% via indirect routes. In children, especially those living near point sources of lead with heavily contaminated soil and dust, the fraction of atmospheric lead uptake through these indirect sources would presumably be much greater.

1. Estimation Strategies and Methods

The numbers of children exposed to lead in dust and soil cannot be separated, as noted above, from the numbers exposed to airborne lead (gasoline or stationary emissions) or leaded paint. Direct exposure to lead, i.e., airborne lead or leaded paint, also foretells simultaneous exposure to dust and soil; therefore, we should sum across estimates for these contributing sources for both potential and actual risk. This summing will lead to overestimates in the numbers of children, clearly making the totals upper bounds. One can determine a lower bound for the estimate by selecting the number of children exposed to the single, main contributor.

An alternative estimating process which avoids double counting, uses multimedia regression analysis. A regression approach, which separates the contributions of lead from various sources to children's blood lead levels, would yield a more precise estimate than the cumulative approach. A regression approach, however, would require (1) establishing a set of regression equations for urban and rural settings in different U.S. regions, (2) determining the prevalence of lead-contaminated dusts and soils in these regions, and (3) using the regression equations and the prevalence data to estimate the number of children exposed to dust and soil lead levels at concentrations large enough to cause adverse health effects.

The general type of regression equation needed simultaneously identifies the independent contributions of leaded paint, airborne lead, lead in dust, and lead in soil to children's blood lead levels. The form of this equation is

$$Y = A_0 + C_1x_1 + C_2x_2 + C_3x_3 + C_4x_4$$

where Y = a child's blood lead level,

A_0 = a constant,

C_{1-4} = the relative contributions of the independent sources of lead,

X_1 = the leaded paint level,

X_2 = the airborne lead level,
X_3 = the level of lead in dust,
X_4 = the lead level in soil, and is a random error term.

Unfortunately, no study to date has produced this type of general regression equation. Available equations either omit leaded paint (Angle et al., 1984; Charney et al., 1980; Walter et al., 1980; Yankel et al., 1977), airborne lead (Charney et al., 1980; Galke et al., 1975), or lead in dust (Galke et al., 1975; Yankel et al., 1977). Some of these omissions reflect regional differences. For example, leaded paint is not an important contributor to children's blood lead levels in the western U.S., and, therefore, it is absent from the multiple linear regression models for children in Idaho (Yankel et al., 1977) and Nebraska (Angle et al., 1984). Other environmental measurement omissions, however, reflect limitations in study design. To successfully use a regression approach for estimating the number of children exposed to hazardous levels of lead in dust and soil would require, at a minimum, an extensive effort in urban and rural areas in the four U.S. regions: West, Midwest, Northeast, and South. The purpose of this effort would be to test children's Pb-B levels and uniformly collect household-specific data on lead levels in paint, air, dust, and soil. These data would form the basis for constructing regional-specific regression equations to predict children's Pb-B levels in urban and rural areas.

A survey of a representative sample of dusts and soils from urban and rural areas of each region is necessary to establish the prevalence of lead-contaminated dusts and soils.

Using the regional- and urban- or rural-specific regression equations, one could determine "safe" lead levels in dust, i.e., levels which do not cause children's Pb-B levels to exceed 25 μg/dl (the present criteria level), or any such level in the future. This determination would be possible by using average lead levels in paint, air, and soil to solve the equation for a "safe" lead level in dust. Similarly, one could solve the equation for a "safe" lead level in soil by using average lead levels in paint, air, and dust.

Once "safe" dust- and soil-lead levels are determined for urban and rural areas in each region, an estimate of the number of children living with higher lead levels of dust and soil can be made. By definition, this estimate would be the number of children exposed to dust and soil lead levels at concentrations sufficient to cause adverse health effects.

2. Results

The numbers of children potentially exposed to lead in dust and soil, but without determining numbers of Pb-B elevations, are taken as the sum of totals exposed to primary contributors to dust and soil:

Paint lead in pre-1940 housing with highest lead content	5.9 Million children
Gasoline lead in 100 of the largest U.S. cities	5.6 Million children
Stationary Source Emissions	0.2 Million Children
Total	11.7 Million Children

As already noted, this total of 11.7 million is an overestimate of undetermined magnitude, since some fraction of children exposed to leaded paint have also been exposed to other sources.

Alternatively, one can select the largest number of children exposed to a single primary source of lead in dust and soil or leaded paint, and consider this number an underestimate. This method may be used because not all children exposed to leaded paint have contact with the other primary generators. This number is 5.9 million children. As an overall estimate, between 5.9 and 11.7 million children are potentially exposed to dust/soil lead.

Estimates of children exposed to lead in dust and soil sufficient to elevate Pb-B levels to potentially toxic ranges cannot be readily obtained in any precise way. One can achieve totals at each Pb-B value, e.g., 15, 20, or 25 μg/dl, for the primary contributors--paint, gasoline, and stationary source emissions as given in earlier sections. A major difficulty for estimating such Pb-B elevations is finding a reliable method for apportioning of a given Pb-B value to either the primary contributor, e.g., paint, or to the receiving pathway, dust and soil.

F. NUMBERS OF CHILDREN EXPOSED TO LEAD IN DRINKING WATER

Studies in the United States and elsewhere have shown that drinking water is a potentially significant source of human lead exposure. Lead can contaminate the water at three points: (1) the water source itself--rivers, reservoirs, and groundwater; (2) the distribution system from water supply to living units, i.e., water mains, and (3) the plumbing in the home, e.g., lead solder. Actually, contamination rarely occurs in water sources from service connection lines and goose necks (connectors for main street to house line), and little is associated with the distribution system. By far, most of the contamination comes from domestic plumbing and plumbing in such public buildings as elementary schools, day-care centers, kindergartens, etc. Specific sources are lead pipe service connections, lead-based solder in copper plumbing, and corrosive (lead-dissolving) water in the plumbing.

While lead in drinking water is usually considered a source in the child's home, a potentially significant exposure risk also exists in such public facilities as elementary schools, kindergartens, day-care centers, etc. One potentially important but little recognized problem in schools and other public facilities is lead contamination of drinking water obtained through taps, water fountains, and coolers. There are several reasons why water in schools could be a hazardous exposure source for young children:

1. Water-use patterns in schools (school periods, weekends, vacations) involve long standing times of water in these units, which permit leaching.
2. Both water cooler-fountains and building plumbing may have lead-soldered joints and other sites of leachable lead, such as lead-containing surfaces in cooling tanks or loose solder fragments in pipes.
3. Unlike the case with lead-containing plumbing in private residences, which affects only the occupants, a single lead-containing cooler-fountain could expose a large number of users.

These findings mean that young children can ingest lead from water at sites other than the home and that the numbers of children and other risk groups exposed to water lead may be expanded to the school-age group.

The magnitude of lead-contaminated drinking water as a public health problem can be seen in the situation that prevailed until recently for half the population of Scotland. The problem was traced to interactions between soft, plumbosolvent water from sources such as Loch Katrine, the city of Glasgow's water supply, and lead pipes and lead-lined tanks in the homes of residents.

Exposure was widespread, and prevalences for elevated Pb-B levels were high. The epidemiology of human lead exposure via drinking water is discussed in detail in EPA's lead criteria document (1986a, Chapter 7 and 11).

In the United States, interactions between drinking water and residential plumbing involve either lead connectors (goosenecks) and service lines (commonly used before 1920) entering the home or copper piping with lead-based solder in the joints--a form of solder that came into use about 1950. Some lead service lines and connectors may have been used after 1920, and lead service connections were occasionally installed until 1986.

Published reports, both in the United States and the United Kingdom, indicate that five major factors contribute to the problem of lead-contaminated drinking water: (1) the length of the time water is in contact with the plumbing--the first water drawn from a standing column of water that has been in contact with lead in plumbing can have a high concentration of lead; (2) water temperature--hot water from pipes containing lead or joined with lead solder lead has more lead than cold water; (3) age of the solder--copper plumbing with lead solder that is less than 5 years old causes higher levels of lead in water than copper plumbing with lead solder that is older; (4) significant lengths of solid lead pipes--water from long sections of lead pipes has high levels of lead; and (5) corrosive water source--all of the above conditions are intensified when the water source is corrosive. The most corrosive water is acidic, soft, or nonalkaline.

Many investigators, both in the United Kingdom and in this country (U.S. EPA, 1986a), have examined the quantitative relationships of Pb-B levels to lead in drinking water. These relationships, reflecting the complex interactions of lead with biological responses, are often described by cube-root and other mathematical functions. Worth et al. (1981) examined residents in Boston, MA, where lead plumbing is still relatively common. These workers found a clear association between lead levels in water and Pb-B levels in children under 6 years old. Children exposed to tap water with lead levels above the U.S. standard of 50 µg/l of water had elevated Pb-B levels, as a group, above 35 µg/dl. These differences were observed during a survey period before corrosion controls were implemented to reduce the lead levels in tap water significantly.

Toxicokinetically, lead in drinking water is probably absorbed more completely than lead in food or other media, especially when the water is drunk

240

between meals or on an empty stomach in the morning. For lead food intake by adults, 10 to 15% of the lead is absorbed, but for water, 35 to 50% or a higher percentage, is absorbed (U.S. EPA, 1986a). Viewed in the context of risk assessment for the case of adults, lead in water presents three to five times the risk for systemic exposure as does lead in food--given the same concentrations of lead. Relative absorption rates are generally higher in children, and the difference may result in rates from water well above the estimate of 50% in children for lead in food (see Chapter III).

1. Estimation Strategies and Methods

As noted earlier, there are three levels of exposure from lead in drinking water that can be defined for U.S. children: potential exposure; some actual exposure at a measurable but not necessarily toxic level; and actual exposure at high toxic risk levels.

In the first approach, estimates of the numbers of children at potential risk of exposure to unhealthy levels of lead from drinking water differ in precision. Several estimates are given below. The estimates of the Division of Housing and Demographic Analysis, Housing and Urban Development (HUD), also include inventories of older housing units (which, as stated earlier, tend to have lead pipe segments at service connections) and provide an index of the persistence of such units in the national housing inventory.

Next considered is an estimation of the numbers of children consuming waterborne lead at some elevated level. According to the analysis by EPA's Office of Policy Planning and Evaluation (U.S. EPA, 1986b), 42 million people in the United States may receive drinking water with lead levels that exceed the proposed EPA maximum contaminant level (MCL) for lead, 20 μg/l, at the tap. With this estimate, one can use Census Bureau data to calculate the number of children at risk for elevated Pb-B levels from water lead levels above 20 μg/l.

EPA's analysis of the extent of lead contamination of tap water uses data on water samples collected by the Culligan water softening company in a cooperative study with EPA. Laboratory analyses were performed by the Illinois Institute of Technology. These 772 grab samples taken at random times during the day were collected in 580 cities in 47 states. They indicate that 16% of the water from U.S. kitchen taps contain 20 μg/l of lead or more. In addition, newly installed plumbing is at particular risk of elevated lead levels because there has not been sufficient time for a film of calcium carbonate to build up on the inside of the pipes, which produces a protective barrier between the water and the materials of the plumbing system. New homes were not included in the Culligan data. Therefore, EPA included the inhabitants of housing built within the past two years to be an additional subpopulation at risk of elevated lead levels in drinking water.

Since this national exposure analysis was completed in 1986, EPA has collected data on local conditions throughout the country. Data on lead leaching rates and contamination patterns in many places confirm that the occurrence of high levels of lead in drinking water is widespread, and may indicate that the national projections underestimate actual exposure (U.S. EPA, 1987c). A significant omission in the national estimate is the prevalence of high lead levels in drinking water in schools.

2. Results

Table VI-12 shows the most general estimates of numbers of U.S. children under 5 years old and those 5 to 13 years old tabulated by age of housing. Using Census Bureau data, U.S. EPA (1987a) estimates that 5.2 million children under 5 years old and 8.7 million children 5 to 13 years old are in older housing, some fraction of which would have old lead service connections for water supply.

The data tabulated in Table VI-12 are of the most general form. A more refined estimation of the numbers of children exposed to water in leaded plumbing is given in Table VI-13, which provides more relevant housing age categories for greatest risk, independent of the corrosivity of water, i.e., homes built before 1920 and the newest units built within the past 2 years. In Table VI-13, the most recent column of age/data is for 1983. In 1983, of the 21 million U.S. children under 6 years of age, 13% or 2.73 million lived in units that had lead water service connections. Similarly, 4% or 840,000 children lived in homes built within the last 2 years, which is the housing fraction having lead-soldered new copper plumbing. Table VI-14, a tabulation from EPA, is similar to that of Table VI-13, but expands the childhood age bands to age 13 and has a 1-year difference in the younger age band.

In Table VI-15, the tabulation shows the relative persistence of aging housing stock. Such data indicate the persistence of the lead service connection problem by virtue of persistence of older housing containing this exposure

TABLE VI-12. NUMBERS OF CHILDREN LIVING IN HOUSING CLASSIFIED BY HOUSING AGE[a]

Age of Housing (% Total)	<5 Years Old	5-13 Years Old	Total
Pre-1940 (29%)	5.2 M	8.7 M	13.9 M
1940 - 1949 (9%)	1.6 M	2.7 M	4.3 M
1950 - 1959 (16%)	2.8 M	4.8 M	7.6 M
1960 - 1969 (20%)	3.6 M	6.0 M	9.6 M
1970 - 1983 (27%)	4.6 M	7.8 M	12.4 M
Total	17.8 M	30.1 M	47.9 M

[a] Source: Statistical Abstracts, 1985: Table 27 (July 1, 1983) and Table 1315 (Fall, 1983).

TABLE VI-13. CHILDREN POTENTIALLY AT RISK FOR LEAD EXPOSURE BY HOUSEHOLD PLUMBING, BY AGE[a,b]

Age of Housing	Number of Children <6 Years 1973	1978	1983	Exposure Profile
Total (Number)	14 M	19 M	21 M	
In housing built:				
Pre-1920 (%)	13	13	13	Lead pipes (+ lead paint)
1920-1949 (%)	25	25	24	Iron pipes (+ lead paint)
1950-1984 (%)	54	55	59	Lead solder (+ lead paint)
Within past 2 years (%)	8	7	4	Fresh lead solder

[a] Source Totals: Special tabulations from 1973-1983 Annual Housing Surveys.

[b] Percentages from Special Report: Division of Housing Demographic Analysis, HUD, Communicated January 7, 1987.

source. For example, about 40% of the oldest, lead service connection (pre-1920) homes are still occupied. This may be an upper-bound estimate because old plumbing has been replaced in some of this old housing stock, but exact information for this is not available.

TABLE VI-14. ESTIMATED NUMBERS OF CHILDREN AT GREATEST RISK OF EXPOSURE TO LEAD IN HOUSEHOLD PLUMBING

New Housing		Population at Risk
8.8 million people in new housing with lead soldered piping[a]:		
(8.8 M)(7.6% of population less than 5 years old)	=	0.7 M
(8.8 M)(12.8% of population 5-13 years old)	=	1.1 M
Total number of children at risk in new housing	=	1.8 M
Old Housing[b,c]		
If one-third of housing units built before 1939 contain lead pipes,[d] then (0.33)(0.29) = 10% of housing have lead pipes.		
(0.10)(17.8 M children less than 5 years old)	=	1.8 M
(0.10)(30.1 M children 5-13 years old)	=	3.0 M
Total number of children at risk in old housing	=	4.8 M

[a] Source: Reducing Lead in Drinking Water: A Benefit Analysis (U.S. EPA, 1986b, based on 9.6 million in new homes and 92% of these homes with metal plumbing).

[b] Source: Derived from Statistical Abstracts, 1985; Table 27, and Table VI-12 of this report.

[c] This group is a subset of the category of children living in housing built before 1939.

[d] Source: David Moore, Office of Policy Development and Research, U.S. HUD, Submissions to ATSDR, January, 1987 and U.S. EPA.

TABLE VI-15. PERSISTENCE OF AGING HOUSING STOCK IN OCCUPIED U.S. HOUSING INVENTORY (MILLIONS)[a]

Cumulative Total Built	Census of 1920	1930	1940	1950	1960	1970	1980
Pre-1920	24		21	20(E)			9(E)
Pre-1930		30	30	29(E)	26(E)		
Pre-1940			35	34(E)	31	27	21
Pre-1950				43	39	36	30

[a] Tabulated values of Division of Housing and Demographic Analysis, HUD: Special Report of January 7, 1987.

(E) = Estimated by authors of HUD report.

Corrosivity of drinking water, i.e., softness, lower pH, etc., is an environmental factor that affects the presence of waterborne lead other than from lead in plumbing. U.S. EPA (1986b) has estimated that about 62 million U.S. people receive such water. If we assume that 11% of these individuals are children under 7 years of age, then about 6.8 million such children are in homes where corrosive water is liable to mobilize lead to some unquantifiable extent.

To estimate the numbers of children exposed to drinking water with sufficiently high lead levels to elevate Pb-B levels, we assumed that children receiving drinking water that exceeds 20 µg/l are at risk of some Pb-B elevation (U.S. EPA, 1986b). A total of 42 million people are estimated to receive water with lead having more than 20 µg/l of water. Census Bureau data indicate that 9% of the U.S. population are children under 6 years of age. Therefore, 3,780,000 children under 6 years of age are exposed to drinking water lead above 20 µg/dl.

In addition to exposure in the home, other sources of water lead exposure may exist during time spent by children in public facilities. Precise numbers of preschool and school age children who may be exposed or are exposed to lead in drinking water in schools, day care centers and other settings cannot now be accurately estimated, given that the necessary survey data are not available. However, exposure to lead in drinking water in these settings can be important. As noted earlier in this section, water use patterns in schools are different from those in homes and they favor the accumulation of leached lead after long lapses in use, for example, summer and holiday break periods and weekends. Such water standing in school plumbing may increase the risk of excessive lead exposure through ingestion of potable water.

With regard to the overall problem of lead in school drinking water, state-level education and health units in two states have reported results from school drinking water lead surveys within their jurisdictions. The Minnesota Department of Health (MDH) recently carried out two surveys of lead in drinking water in Minnesota schools. The first and smaller survey (Minnesota Department of Health, 1986) consisted of two phases. In Phase I, 31 schools in 30 Minnesota cities were surveyed, including 24 elementary schools or schools with elementary grades. Of the 67 water samples collected and analyzed, 17 (25%) exceeded the current EPA limit of 50 µg/l and 27 (40%) exceeded the proposed goal of 20 µg/l. These were "first-flush" samples obtained after weekend or holiday periods. In Phase II, samples at five of the original sites were examined for both first-flush and post-flushing lead levels. First-flush levels ranged from 2.2 to 500 µg/l with a mean of 141 µg/l (median = 38 µg/l). After 1-minute flushing, the mean value was 9.5 µg/l (median = 6.7 µg/l).

The first survey was followed up with a much larger statewide survey, which has just been completed. Preliminary summary statistics provided by the MDH indicate that two first flush samplings, 30 days apart, were obtained from 104 (25%) of Minnesota's 414 school districts. These repeat samplings consisted of 1,157 and 1,051 water samples respectively. In the first data set, 74 samples (6.4%) exceeded the existing EPA limit of 50 µg/l and 166 samples (14.3%) exceeded the proposed 20 µg/l goal. For the second sampling, the corresponding exceedences were 85 (8.1%) and 150 (14.3%), respectively. In neither survey was the nature of the tap water drinking source specified, e.g., non-refrigerating fountain, electric cooler, or other sources.

In a similar type of survey, the Maryland Department of Health and Mental Hygiene (MDHMH) examined drinking water lead levels in three stages in the summer of 1986, as described in Maryland Department of Health and Mental Hygiene news releases (September 22, 1986 and September 30, 1986). These schools were characterized as buildings less than four years old or units where new plumbing was installed in the last four years. Lead leaching was such that flushed water lines began to accumulate the toxicant after two hours of standing without further use. In July 1986, a survey of 45 schools, including a number of schools in the city of Baltimore, showed that 20 of them (44%) had lead levels above the EPA limit of 50 µg/l. In September 1986, MDHMH reported that a second survey yielded 33 (30.5%) of 108 schools with lead levels exceeding the EPA limit of 50 µg/l. Further examination of MDHMH's summary data as provided in the releases indicates that water samples from 72 (67%) of these 108 schools were at or above the proposed EPA goal of 20 µg/l. In a third stage of the Maryland school survey, carried out in September 1986, 30 schools were examined for the first time, and two of them exceeded the 50 µg/l limit. However, examination of the MDHMH summary data indicates that 9 (30%) of the schools were at or above the proposed goal of 20 µg/l. From the available information it appears that the new lead soldering in school plumbing and/or lead contamination in fountains themselves are suspected sites of lead leaching into water.

Contamination of drinking water by lead in other public facilities has also been reported. Unpublished preliminary data on lead levels in water from drinking fountains/coolers at two U.S. Naval facilities have been made available to the U.S. EPA and ATSDR (see Table VI-16). Of the approximately 90 fountains/coolers sampled by the Navy contract laboratories, water lead levels above the current EPA 50 µg/l limit were found in about 35% of combined first-flow, 1-minute, and 2-minute draw samples. Of these fountains/coolers, over 60 were major brands; the remainder were not identified by brand. In this survey "first-flow" refers to the first water obtained from the source at various times during the day. Thus, such samples may not have been the first flush of water after overnight standing. Of 39 first-flow samples, 23 (58%) were at or above a proposed EPA goal of 20 µg/l, and 31 (72%) were at or exceeded the level of 10 µg/l. For the 1-minute draw samples, 26 (72%) were at or above 20 µg/l, while 30 (83%) were at or above 10 µg/l. While it is not fully clear how much of the lead in these samples was contributed by the fountains/coolers and how much by the buildings' plumbing, water from some fountains/coolers in the Navy survey contained additional lead above that expected based on water lead levels from plumbing lines or taps in the same building.

TABLE VI-16. LEAD LEVELS IN WATER SAMPLES OBTAINED FROM WATER FOUNTAINS/COOLERS AT NAVAL FACILITIES IN MARYLAND[a]

Parameter	Lead in First Flow	Parameter	Lead in 1 or 2 Minute Flush
	N = 39		N = 90
High	570 µg/l	High	830 µg/l
Mean	101 µg/l	Mean	69 µg/l
Median	39 µg/l	Median	30 µg/l
Low	<5 µg/l[b]	Low	<5 µg/l[b]

[a] Source: Based on data obtained from U.S. Navy (1987).

[b] Analytical detection limit of 5 µg/l.

The above data from two separate states' surveys of schools and from Naval facilities in Maryland raise important questions about the potential for significant lead exposure from sources of potable water in public facilities. Systematic evaluation of this potential problem will be needed to determine its scope and any appropriate corrective measures.

Table VI-17 shows EPA's preliminary calculations of the relationship of tap-water lead in homes as a function of both pH and age of house. As expected, first-flush samples are more apt to have excessive lead concentrations than fully flushed collections. The more acid, i.e., more corrosive, water produces the greater lead contamination at values above 20 μg/l, the EPA proposed standard. Although the frequency of elevated lead samples decreases with the age of the home, there are still unacceptable first-flush percentages for corrosive and neutral waters, i.e., 51% and 14%, respectively, in houses 6 years and older. Equally important is the finding that in houses up to 2 years old that have corrosive water with a pH ≦6.4, 51% of the samples of fully flushed water contained more than 20 μg/l of lead.

TABLE VI-17. PERCENTAGE OF VARIABLY COLLECTED WATER SAMPLES EXCEEDING 20 μg/l OF LEAD AT DIFFERENT pH LEVELS AND BY AGE OF HOUSE[a]

		Percent of Samples >20 μg/l	
Age of House	pH	First Flush	Fully Flushed (2 min)
0-2 years	≦6.4	93	51
	7.0 - 7.4	83	5
	≧8.0	72	0
2-5 years	≦6.4	84	19
	7.0 - 7.4	28	7
	≧8.0	18	4
6+ years	≦6.4	51	4
	7.0 - 7.4	14	0
	≧8.0	13	3

[a] Source: U.S. EPA Office of Drinking Water (1987c); preliminary results from "Lead Solder Aging Study."

Estimations for the category of actual exposure to water lead sufficient to cause Pb-B levels with toxicity risk are those employed by U.S. EPA (1986b) in its examination of numbers of U.S. children who would be above certain criterion values because of drinking water lead levels above 20 μg/l. U.S. EPA (1986b) employed logistic regression analyses techniques analogous to those employed for projections of children whose Pb-B levels would decline from specified Pb-B levels from phasedown of gasoline lead (U.S. EPA, 1985).

In this analysis of the benefits of reducing the lead standard for drinking water, U.S. EPA (1986b) estimated that 241,100 children had Pb-B levels above 15 µg/dl due to lead in their water as a result of the action of corrosive water on aged plumbing. Of these, EPA estimated that 100 children have Pb-B >50 µg/dl as a result of waterborne lead, 11,000 have Pb-B levels between 30 and 50 µg/dl, and 230,000 have Pb-B levels between 15 and 30 µg/dl.

G. NUMBERS OF CHILDREN EXPOSED TO LEAD IN FOOD

Dietary lead can account for a significant portion of the total body lead burden in populations not having sizable exposures to the sources already discussed. In many people, it can add enough to cause an elevation in Pb-B levels. Dietary lead intake is important because it is a source of exposure for the entire population. Since food is ingested in relatively large amounts, lead concentrations in food at the parts-per-billion (ppb) level correspond to intakes of microgram quantities. For example, an average food content of 50 ppb of lead yields an intake of 50 µg/day when 1 kg of food is eaten. These 50 µg will elevate the Pb-B level by a measurable amount, about 8 µg/dl, using one relationship cited in U.S. EPA (1986b).

Over the centuries, lead in food and beverages has provided much of the toxicologic record for lead poisoning, especially when lead was used in large amounts as an adulterant (see, for example, Wedeen, 1984). At present, lead enters food at production, harvesting, processing, and distribution steps (U.S. EPA, 1986a). In addition, beverages and other liquids can be contaminated by improperly glazed pottery (Klein et al., 1970) and various utensils.

We cannot determine how much of the total food lead is due to human activity without knowing the background level. Wolnik et al. (1983) gathered background or near-background levels in cereals, grains, vegetables, and several meats. When we relate the results from these studies to current food consumption surveys conducted by the FDA, we find that production and processing increase the lead content of food 2-fold to 12-fold. Sources of lead at the production stage include fallout onto plants, some lead uptake through the root system, and lead in forage and soil in areas where livestock graze. Lead enters in food processing mainly via lead-soldered cans.

In the late 1970s, the use of lead to solder the seams in cans began to be phased out, and consequently, the lead in canned food has significantly decreased. With infant foods, for example, lead levels in evaporated milk have declined, on the average, from 0.5 µg/g wet weight in the early 1970s to 0.07 µg/g in 1981. Similarly, lead levels in some juices have declined about 95%.

The daily dietary intake of lead in young children has been examined in several surveys. Beloian (1982, 1985) has proposed and used a food consumption model for evaluating daily contaminant intake. The elements of this model include the numbers of times specific foods are consumed in 14 days, lead content, and size of portions. Three age groups of children were examined: 0 to 5 months, 6 to 23 months, and 2 to 5 years. Lead levels in food groups were averaged over the period 1973-1978.

Table VI-18 shows data reported by Beloian (1982). They include the mean daily lead intakes and the distributions of lead intake by indicated percentiles, with food lead data for 1973-1978. Mean values are moderate, but at the higher percentiles the lead intakes are sizable, which emphasizes the importance of considering distribution phenomena. These numbers are based on older, higher food lead measures than may exist currently.

TABLE VI-18. DAILY MEAN DIETARY LEAD INTAKE BY PERCENTILES[a]

Age Group	Mean Intake (µg/day)	Percentiles			
		50	90	95	99
0-5 months	15	11	31	36	55
6-23 months	59	54	89	110	140
2-5 years	82	79	120	130	170

[a] Adapted from Beloian (1982). Based on averaged data from 1973-1978.

Bander et al. (1983) conducted a nationwide 7-day food consumption survey of 371 preschool children 0 to 5 years old, using food intake levels provided by the National Food Processors' Association in 1980. Table VI-19 shows the means of lead intake, the means related to kilocalories, and the means per unit mass of food as a function of age.

In the Beloian (1982) study, Table VI-18, the three age groups had mean intakes of 15, 59, and 82 μg/day. The corresponding lead intakes expressed as a function of body weight (kg) were 2.7, 6.1, and 5.6 μg/kg.

Bander et al. (1983) found that children had a mean intake of 62 μg/day lead on a subject basis and 22 μg/day on a caloric or food mass basis. Partitioned by age, the intake ranged from 49 μg/day for infants to 74 μg/day for 5-year-olds (Table VI-19). Because of the continual reduction in the lead content of foods, estimates of current dietary lead intake are smaller.

TABLE VI-19. MEAN DAILY DIETARY LEAD INTAKE IN PRESCHOOLERS CLASSIFIED AS TOTAL OR NORMALIZED DAILY INTAKE[a]

Age (years)	Total Mean (μg/day)	Mean/500 kcal	Mean/500 g Food
<1	49	17	16
1	55	24	21
2	56	22	20
3	65	22	22
4	65	22	21
5	74	20	22

[a] Adapted from Bander et al. (1983). Based on food lead measurements in 1980.

1. Estimation Strategies and Methods

The number of persons potentially exposed to some level of lead in food includes the entire U.S. population because a centralized food production and distribution system serves virtually all parts of the nation. Each food processing step adds to the amount of lead in food (U.S. EPA, 1986a). Therefore, all U.S. children under the age of 6 are at potential risk. We calculated the number for the 1985 estimated population, using the lowest projection method and found 9% of the total population to be under 6 years of age. This amounts to about 21 million children.

We used the following strategy to evaluate the number of children exposed to lead levels in food that are high enough to elevate the Pb-B so that it approaches some toxicity level:

(1) The Pb-B level associated with lead in food should not be more than 10 μg/dl, given other inputs such as from drinking water, contact with dust and soil, and even direct inhalation--all of which also contribute to

Pb-B and push the final level unacceptably close to 20 to 25 μg/dl, the lead toxicity risk levels identified by the CDC (1985) and WHO (1986).

(2) A relationship of lead in food to Pb-B in infants and toddlers derived from published data (Ryu et al. (1983) is Pb-B (μg/dl) = 0.16 x diet Pb/day (μg/day). From this relationship, a Pb-B less than or equal to 10 μg/dl from food requires a lead in food intake of less than or equal to 62.5 μg/day.

(3) The percentage of children who have lead intake at or above about 65 μg Pb/day should then be selected from those studies whose results can be applied to the nation as a whole.

Surveys by Beloian (1982) and Bander et al. (1983) were designed so that they can be applied nationally for the years when the lead levels in foods were measured. Given the centralized food system, any comprehensive U.S. survey of lead levels in food becomes a national survey.

The Beloian (1982) data, Table VI-18, show that the 95th percentiles of daily lead intake are 36, 110, and 130 μg/day for children aged 0 to 5 months, 6 to 23 months, and 2 to 5 years, respectively. If overall declines for average lead levels in food are assumed to be at least 50% from 1973-1978 to the present and across the entire distribution, these levels would now be 18, 55, and 65 μg/day. Uniform downward changes across the entire distribution are reasonable, assuming a centralized food supply and no major changes in general food intake habits from 1973 to the present. The last value, about 65 μg/day, is the upper limit of lead levels in food to produce Pb-B levels lower than 10 μg/dl. For the 6 to 23 months group, 55 μg/day is also close enough to the limit of concern for the 95th percentile. While we are not sure about the relative vulnerability of the very young (0 to 5 months old) to lead exposure, this youngest group has a very low intake and is not included in the analysis. This approach allows us to say that, at most, 5% of the children under 6 years of age, excluding children 0 to 5 months old, are at or approaching a dietary lead exposure that pushes their body burden close to that associated with early toxicity if they are also exposed to other typical lead sources.

In general, the Belonian percentiles agree with other data. Bander et al. (1983) noted that 8.9% of children had daily lead intakes of at least 100 μg/day. Most of these children were 4 to 5 years old. Considering that this study was conducted more recently and used food processors' data for 1980 analyses and the lower levels of lead at the 95th percentile for younger children, the Bander et al. (1983) results are consistent with those of Beloian (1982).

Uncertainties are inherent in these approaches for estimating the numbers of children who are exposed to lead in food to a degree sufficient to cause measurable elevations in Pb-B levels. First, the percentage of decline in food lead from 1973-1978 to more recent periods is difficult to determine. We cannot specifically compare data gathered before 1981-1982 with more recent survey findings. The lead content of certain categories of foods has been markedly reduced (see Chapter IX), but changes in the overall dietary intake of lead are difficult to measure. Second, the direct/indirect air lead contributions are difficult to measure. Third, the criterion Pb-B value selected for calculating background levels is not a precise measure. Finally, selecting a single value for lead intake in food about a given percentile affects the margin of toxicological safety. If the current value is significantly below about 63 μg/day at the 95th percentile of the distribution then the resulting Pb-B will be lower, and the other levels must be adjusted accordingly.

2. Results

The number of children under 6 years is estimated to be 21,405,000 (World Alamanac, 1987); this number is based on the Census Bureau projections for 1985 by the lowest of three methods. Since we exclude infants 0 to 5 months old from our calculations, the base population is reduced to about 19,474,000.

Of this estimated number, a maximum 5%, or 973,700 children under 6 years old, receive enough lead from food alone to constitute a potentially unacceptable lead burden, as indicated by Pb-B levels. If the increase in Pb-B from food is lower than the one selected here (i.e., less than 10 μg/dl), then the numbers of children at risk would be lower for a given daily lead intake. On the other hand, the reference Pb-B level of 20 μg/dl selected for the estimate is not the lowest criterion level for the purpose.

H. SUMMARY AND OVERVIEW

In this chapter, estimates of the numbers of lead-exposed young children, arranged by the source of exposure, are derived and described. These estimates define different degrees of exposure, both within an exposure category and across exposure categories. Overall, the data sets used for these source-based analyses vary highly in their precision and accuracy, in their assessment and definition of actual versus potential exposure, in their representativeness for childhood exposure nationwide, and, finally, in the relationship between the degree of exposure and some toxicity risk.

With regard to the accuracy and precision of numbers relative to source of lead, it is not possible to measure the level of estimation error for each of the source categories. For example, in some cases we deal with actual counts of individuals, while in other cases we are confined to combining various elements of an estimation analysis, each having variable and relatively undefined precision. Some estimates are also judged to be upper or lower bounds for actual values.

In a number of the estimation procedures, available data for the prevalence of elevated Pb-B levels among children are derived from either regional analyses or reasonably matched strata of children from NHANES II, and their representativeness for the actual census of source-exposed children must be carefully appraised.

Finally, the various source-based estimates of actual exposure to lead will differ as to how closely measures such as blood lead can be related to what we term some toxicity risk. In some cases, a Pb-B criterion value associated with presently understood toxicity risk is used, while in others we can only say that a blood lead elevation is predicted to occur at some undefined upper value. It should be noted that definitions of toxicity in terms of Pb-B values have been declining. For this reason, multiple Pb-B values are used where calculable. For purposes of summary and discussion, each source-based estimation analysis is treated separately.

1. Paint Lead as an Exposure Source

We have presented three levels of estimates for young children exposed to lead in paint. The first two analyses involve actual U.S. Census and housing counts and can be taken as reasonably accurate enumerations of children who are at least potentially, if not actually, exposed to lead in paint.

The most general approach was to determine U.S. Census enumerations of young children (less than 7 years old) and then compile which portion of those children will be in residential units having leaded paint meeting a minimal definition of exposure: greater than or equal to 0.7 mg/cm^2. In so doing, we determined that the best available data for this analysis provided a "national best estimate" of about 12 million children potentially exposed to lead in paint, with about 13.6 million children for the upper bound. Of these, about 5.9 million children are in pre-1940 residential units. In this case, we did not address leaded paint in the presence of those factors that might additionally enhance childhood lead paint exposure, for example, deterioration indexed by peeling leaded paint, lead-painted broken plaster, etc. Such indices of deterioration are not required before leaded paint exposure becomes a problem. For example, children can readily gnaw and chew on lead-painted woodwork such as window sills, even if the painted surfaces are in good repair.

The next level estimated involved enumeration of children determined to be residing in unsound/substandard housing. Here we would expect that the probability for lead exposure sufficient to elevate blood lead to some level would be much higher than it would be through a simple housing count, where undeteriorated and deteriorated housing is combined. Using figures from one study, the best national and upper-bound estimates for the numbers of children in such lead-painted, unsound residential units are about 1.8 and 2.0 million children, respectively. These figures are probably an overestimate since units having one characteristic of disrepair would also have others, yielding double counting in at least some cases.

These figures complement the numbers of children presented as census data enumerations by SMSA presented in Chapter V, Section C. For example, the total census count of children in the 318 SMSAs who live in residential units built before 1950 amounts to about 4.4 million (Table V-20). From Table VI-3, the number of children (national best estimate) in all pre-1940 housing is approximately 5.9 million. Taking into account all factors of differences in the two analyses, approximately 75 to 80% of all children in older U.S. housing having leaded paint are found in urban areas.

The numbers of children in unsound housing tabulated in this chapter do not include those children of families who are now moving back to older housing in cities and rehabilitating such housing as part of the phenomenon of gentrification. This number may be sizable but it is not possible to estimate such a subset of the urban child population.

To examine the number of children estimated to have actual lead exposure sufficient to cause Pb-B elevation, we have employed prevalences of different Pb-B criterion values for children in inner-city housing and derived from either a survey of Chicago housing (Pb-B >30 µg/dl) or NHANES II projected prevalences at Pb-B levels >15, >20, and >25 µg/dl. The Chicago-based prevalence of 12.8% applied to children in deteriorated housing yields a total of approximately 230,000 children with Pb-Bs above 30 µg/dl. The numbers of children in such housing with Pb-B levels above 15, 20, and 25 µg/dl were, respectively, about 1.20 million (15 µg/dl), 0.55 million (20 µg/dl), and 0.19 million (25 µg/dl). Although we recognize that leaded paint in elementary schools, kindergartens, etc., will pose potential risk for young children, we cannot as yet quantify this segment of the leaded paint problem. Similarly, leaded paint inputs to dusts and soils around public facilities are not readily quantifiable.

2. Gasoline Lead as an Exposure Source

Estimating the numbers of young children with potential exposure to lead from combustion of leaded gasoline is not a straightforward process. In this report, an estimate was based on the numbers of children living in the largest U.S. urban areas where vehicular traffic is expected to figure significantly in childhood exposure. For the largest 100 U.S. cities and for children less than 7 years of age, this figure is approximately 5.6 million children.

To examine numbers of children having exposure to gasoline lead at various Pb-B criterion values, this report relied on data from EPA's Office of Policy Analysis. The EPA report derived numbers of children predicted to fall below indicated Pb-B values with the phasedown of lead in gasoline, and projections were extended to years beyond 1987. For 1987, for example, 563,000 children up to 13 years old will have Pb-B declines to below 20 µg/dl. The corresponding number for a Pb-B level of 15 µg/dl was approximately 1.6 million. Please note that in this case the age interval for defining children extends to 13 years of age. It is not possible to adjust these figures to the age interval up to 5 or 6 years employed in most cases, since Pb-B distributions are child age-dependent and do not permit a simple linear fractionation of these figures.

Estimates provided by EPA do not reveal specific Pb-B values for the children in the analysis, but rather shifts in Pb-B levels sufficient to cause declines below Pb-B criterion values are shown. With data displayed in this manner, the effect of gasoline lead reduction is focused on relative toxicity risk, that is, the Pb-B criterion values.

3. Lead From Stationary Sites as an Exposure Source

The numbers of children exposed to lead from stationary source emissions have not been well catalogued and related to potential exposure risk. One study found that approximately 230,000 children are in the exposure zones associated with operations such as smelters, refineries, acid-lead battery plants, etc.

A range of estimates for the number of children actually exposed to lead from such sources was also presented where prevalences were restricted to results from several specific smelters. A likely national best estimate could not be derived from the limited data. For primary smelter operations, prevalences range from 1 to 26% (Pb-B ≥25 μg/dl and ≥35 μg/dl EP). The corresponding figure for secondary smelters, based on one survey, is 4% (Pb-B >20 μg/dl). The corresponding numbers for primary operation communities range from 210 to around 5,500 children. With secondary smelters, the number is approximately 7,500 children.

4. Lead in Dust and Soils as an Exposure Source

To estimate the number of young children exposed to lead in dust and soil, we combined the numbers of children exposed to the primary generators of this source category, i.e., lead in paint and lead from stationary/mobile (gasoline) lead sources. As described in the report, the upper limit of young children potentially exposed in this category is approximately 11.7 million and the lower limit is 5.9 million. The number of children actually exposed to dust/soil lead levels sufficient to cause elevated Pb-B levels that are also distinguishable from elevations due to paint, etc., cannot be easily determined by the various methods for obtaining estimates.

5. Lead in Drinking Water as an Exposure Source

One estimation method shows that approximately 1.8 million children less than 5 years old and 3.0 million children 5 to 13 years old have potential risk for lead exposure from parts of old residential plumbing. For new homes with new and leachable plumbing lead solder, the corresponding numbers are calculated as 0.7 million and 1.1 million, respectively.

Next, the estimate for numbers of children having drinking water lead exposure sufficient to cause some Pb-B elevation but not necessarily to toxic levels is given. As derived from EPA's statistics, 20% of public drinking water supplies exceed the proposed Maximum Contaminant Level (MCL) of 20 μg/l, which eventually yields a total of about 3.8 million children. These children will have Pb-B elevations calculated as being at or above 3 to 6 μg/dl, based on the relationship between ingested lead and Pb-B [Pb-B = 0.16 x μg Pb/day (from water)] with daily intake of 1 or 2 liters of water at or above 20 μg/l. At this time, we estimate that 241,000 of these 3.8 million children would have a Pb-B level above 15 μg/dl, with 230,000 between 15 and 30 μg/dl, 11,000 over 30 μg/dl, and 100 over 50 μg/dl.

6. Lead in Food as an Exposure Source

When we examine this category of source-specific lead exposure in young children, we not only have a large affected population base for potential exposure, but one that is the focus of much activity to reduce the lead in this medium.

With respect to potential lead exposure from food at even some modest level of contact, virtually all young children will ingest some measurable amount of lead: that is 21 million children. The children estimated to have a lead exposure risk sufficient to cause some rise in Pb-B is approximately 5% of this base population, about 1 million children. This number is based on both lead levels in foods measured in the 1970s and on those levels adjusted for declines in more recent times; therefore, it may be an overestimate to some extent.

7. Ranking of Lead-Exposed Children by Source

It is not possible to rank rigidly the numbers of lead-exposed children from each lead source, a difficulty that applies to both potential and predicted actual lead exposure. For health assessment, it is more useful to consider the overall impact of each category and to rank qualitatively their specific characteristics. The reasons for this are related to the nature of the different estimation approaches, the precision of the estimation process, and the definitions of potential and actual lead exposure and the size of the age intervals employed in source-specific estimating.

When specifically estimating actual lead exposure, any rigid ranking of children exposed by source can be misinterpreted with underestimates of particular concern. A good example of this is the data for children exposed to lead in paint at levels that elevate Pb-B to the toxic range where at least two factors would militate in the calculation of a low estimate.

We can provide the following general findings and conclusions about lead sources for childhood exposure and in utero exposure.

- In terms of both quantitative impact and persistence of the hazard, as well as dispersal of the source into the population, leaded paint has been and remains a major source for childhood exposure and intoxication.
- Following close to leaded paint as a troublesome and persistent lead source is dust/soil lead, dispersed over huge areas of the nation.
- Drinking water lead is now recognized as a potentially significant exposure source in both the home and in schools and other public facilities; a particular hazard is electric water coolers, as documented in the section on drinking water.
- From examining the above lead sources, they are all related, collectively as multi-source exposure in old housing and particularly old housing in varying stages of disrepair.
- Gasoline lead is declining significantly as a major lead source, particularly since the 1970s when it was adding about 40 to 50% to total Pb-B levels in the U.S. population.
- Stationary sources provide a very limited, though potentially high, source of lead exposure.
- Lead in food is declining in importance as a general exposure source, but daily intakes for recent years are still enough to add measurable amounts to total Pb-B levels of children.
- Time did not permit the detailed quantification of fetal exposure, via pregnant women as the surrogate risk group, in terms of source-specific exposure.
- With pregnant women, lead from food and water would be the main contributors to Pb-B levels above those considered "safe" for fetal protection.

- Since food and water lead primarily produce the projected Pb-B levels and total exposure counts for the four pregnancy categories in Chapter VII, we can estimate the joint contribution of food plus water to produce these numbers.

VII. EXAMINATION OF NUMBERS OF LEAD-EXPOSED WOMEN OF CHILDBEARING AGE AND PREGNANT WOMEN

In pregnant women, lead readily crosses the placental barrier early in gestation (see Chapter III). *In utero* exposure therefore occurs at periods of embryological development when important organ and system elaboration can be affected adversely by lead uptake. Such adverse *in utero* effects have been known for many years and low-level lead effects remain a public health problem. These effects were documented earlier in this report, in Chapter IV, and by such critical assessments as EPA's lead criteria document (U.S. EPA, 1986a).

Two important points can be made now concerning *in utero* lead exposure: *in utero* impact can be irreversible and the adverse impacts of maternal blood lead have been found at low levels, between 7 and 17 µg/dl, based on current studies (see Chapters III and IV). Pregnant women are recognized as a high risk population segment because of *in utero* exposure of the fetus. These risk definitions imply that every pregnancy potentially represents a fetus at risk if the mother has a blood lead level of 10 µg/dl or higher. Since the composition of pregnant women is not a predictable segment of the population, women of childbearing age are also examined in this chapter.

A. STRATEGIES AND METHODS

We have first established numbers of women of childbearing age and pregnant women by two race and two age groups. We decided to examine women residing in SMSAs and of childbearing age for 1984, a relatively recent date that matches our examination of young children, and for which relevant Pb-B prevalences could be estimated. In addition, estimates of pregnant women for that year were obtained to illustrate the extent of risk to fetuses at any given time.

1. Methods

The following specific steps were taken to establish the required information for the populations and estimates of prevalences of blood lead levels among them:

1. U.S. Census Bureau population projections for 1984 were used, as well as 1980 census data on residential distribution, to estimate the number of white and black women aged 15 to 19, and 20 to 44 years of age and who live in SMSAs.
2. From the Division of Vital Statistics of the National Center for Health Statistics data, the numbers of live births occurring in SMSAs in 1984 were counted, and fetal deaths for 1984 apportioned to SMSAs. Data for legal abortions for 1984 are not yet available, and other data from CDC (1983, 1986c) were utilized. The number of pregnancies regardless of outcome were estimated for the four race/age strata of women in SMSAs.
3. Estimates of prevalences of Pb-B levels of interest in pregnant women for 1984 were provided by the U.S. EPA Office of Policy Analysis, Washington, D.C., (J. Schwartz and H. Pitcher) using the same methodology as cited to project Pb-B level prevalences in young children for 1984. The Pb-B criterion values of concern were >10, >15, >20, and >25 µg/dl. The estimated prevalences for use in 1984 were necessitated by the observed declines occurring in Pb-B from the time of the NHANES II survey period to more recent years.

With respect to the outline of approaches employed in this chapter, several points require discussion. The selection of the two age categories for women of childbearing age is based on the fact that women below 20 years of age tend to have a high risk of pregnancies with poor outcomes in general and without specific reference to the blood lead status of the mother (National Research Council, 1987). Further, the NHANES II data for women of childbearing age indicate not only that blood lead levels showed variations for white and black women, but that women of either race showed variation by age. We therefore examined four categories in this population: white, aged 15 to 19; white, aged 20 to 44; black, aged 15 to 19; black, aged 20 to 44.

The attempt to estimate all pregnancies regardless of outcome is based on the recognition that the various outcomes are not intrinsically relevant to the risk of fetal exposure to maternal blood lead levels. Legal abortions were included since data are available for the extent of this outcome; fetal wastage, that is, spontaneous abortions before 20 weeks of gestation, were not considered since no data exist.

The estimate of women of childbearing age includes some proportion of women who will never experience pregnancy. We know of no method to estimate this proportion. However, we believe that consideration of the number of pregnancies in a given year provides some measure of assessing the size of the surrogate population at risk.

The Census Bureau projects future population utilizing three methods of calculation. The so-called middle series projections for 1984 were used (Table 6, U.S. Bureau of the Census, 1984), which provide estimates by age, sex, and race, and have a 12% error for the 1984 projections (Table N, U.S. Bureau of the Census, 1984). These are projections for the entire country, and to establish the proportion of women living in SMSAs, the 1980 residential distribution was applied to the projected 1984 figures for the four categories of women (U.S. Bureau of the Census, 1983).

The data available for 1984 live births consisted of computer output (Division of Vital Statistics, National Center for Health Statistics) from which we had established the number of live births for each SMSA as defined in 1980. The births were identified by race, but not by age of the mother. We used the latest available data, 1981, for distribution of live births for each race by the mother's age (USDHHS, 1985a) to allocate the 1984 live births for all SMSAs. The fetal deaths for 1984 were provided by the Division of Vital Statistics and were allocated to the maternal age categories within race groups by applying published information for 1981 (USDHHS, 1986).

Information for 1984 legal abortions is not yet available, and we utilized 1983 data. Examining the data available for the years 1971 through 1983, a peak in rates appears in 1980 and since then rates have declined very gradually. Since the number of women of childbearing age has increased steadily, the actual number of legal abortions continued to increase through 1982. In 1983, not only the rates but the actual number of abortions showed a decrease. Since the decrease of the rates is quite gradual and the actual number for 1983 below those for 1980, 1981, and 1982, employing the 1983 rates for 1984 seemed conservative.

CDC data concerning legal abortions provides ratios of abortions to live births (per 1,000 live births) and a rate of abortions (per 1,000 women aged 15 to 44) for each of the states and the District of Columbia. A national ratio and rate are also presented. Information on the distribution of abortions by age and race are also provided, but these data are not available on a state basis.

We used two methods of estimating 1984 abortions for the SMSAs and used the result that provided the smaller numbers. The discarded method consisted of applying the ratio for each state to the number of live births that had occurred in SMSAs in that state in 1984. The sum of these calculations was then proportioned into race and age categories according to data available for 1983. The alternate method consisted of applying the rate (per 1,000 women 15 to 44) to the 1984 population estimates obtained from the Census Bureau projections for 1984. The resulting total was allocated to the race/age categories according to 1983 abortion data. A difference of about 3% was observed between the two methods and the smaller total was selected for inclusion.

The NHANES II prevalence data base, 1976-1980, for distributions of Pb-B levels in the population, was used to estimate prevalences for Pb-B criterion values for 1984, using logistic regression techniques to adjust the original Pb-B prevalences to 1984. Adjustment was necessary for the reasons discussed in Chapter V and Appendix G. The estimated prevalences were calculated only for women of childbearing age residing in SMSAs. The prevalence rates shown in Table VII-1 should be considered while bearing in mind that the criterion value of 10 µg/dl lies in a very narrow portion of the Pb-B range. Certain prevalence values in the table, particularly that for the older group of black women, appear to be unusually high when compared to the other prevalences. However, because of the narrowness of the Pb-B range at 10 µg/dl, rather small changes in the mean Pb-B values will account for rather large differences in prevalences. The geometric mean shown for older black women is 7.3 µg/dl, compared to geometric means of 3.4, 5.2, and 5.1 µg/dl for the other three groups.

The pattern of increasing group values with increasing age, discussed in Chapter 10 of the EPA lead criteria document (1986a), can be seen in the means for each of the two racial groups. U.S. EPA (1986a) reports an increase of

TABLE VII-1. ESTIMATED PERCENTAGES OF WOMEN OF CHILDBEARING AGE EXCEEDING SELECTED Pb-B VALUES, THEIR GEOMETRIC MEANS AND STANDARD DEVIATIONS (μg/dl) BY RACE AND AGE, FOR POPULATIONS IN ALL SMSAs, 1984[a]

Race/Age	(Yrs)	Pb-B (μg/dl)				Geometric Mean	Geometric SD
		>10	>15	>20	>25		
White	15-19	9.2	0.5	0.1	0.03	3.4	1.78
	20-44	9.7	1.8	0.4	0.1	5.2	1.65
Black	15-19	8.2	1.3	0.2	0.05	5.1	1.62
	20-44	19.7	3.7	0.7	0.2	7.3	1.54

[a] Estimates of prevalences provided by J. Schwartz and H. Pitcher, U.S. EPA Office of Policy Analysis, Washington, D.C.

approximately one microgram for each decade of age increase. The differences in geometric means for young and older women in each of the racial groups amounts to about 2 μg/dl. The prevalence estimates were then applied to the estimated population strata, and the findings are presented in Table VII-2.

2. Results

Census projections for 1984 and demographic distributions for 1980 were utilized to estimate the 41,300,000 white and black women of childbearing age who lived in SMSAs in 1984. The estimated numbers of these women in the four race/age categories above the selected Pb-B levels were: >10 μg/dl, 4,460,600; >15 μg/dl, 761,400; >20 μg/dl, 161,600; and >25 μg/dl, 41,800.

The estimating procedure outlined above yielded a total of 3,595,000 pregnant women for 1984. Of these, 403,200 are estimated to have a Pb-B level above 10 μg/dl, 69,400 above 15 μg/dl, 14,500 above 20 μg/dl, and 3,800 above 25 μg/dl.

Estimated prevalences of Pb-B at these selected levels for 1984 are lower than those obtained from the survey data collected during 1976-1980, which is attributable to the reduction in ambient air lead pollution, (see Chapter IX). However, recall that women have a smaller uptake of airborne lead than children on a body weight basis. Unlike children, they obtain the major portion of the total body burden of lead from food and water; a smaller fraction is derived from paint, dust, and soil lead. Lead in the food of adults, providing exposure for teenage and adult women, may not be reduced by the same amounts as for infants and toddlers (see Chapter IX), although reductions are certainly occurring across all age groups.

TABLE VII-2. ESTIMATED NUMBER OF WOMEN OF CHILDBEARING AGE AND ESTIMATED NUMBER OF PREGNANT WOMEN AND PROJECTED NUMBERS ABOVE FOUR SELECTED Pb-B CRITERION VALUES (μg/dl), BY RACE AND AGE, IN ALL SMSAs, 1984

Race/Age	(Yrs)	Number	Pb-B (μg/dl) >10	>15	>20	>25
Women in SMSAs[a]						
White	15-19	5,478,000	504,000	27,400	5,500	1,600
	20-44	29,740,000	2,884,800	535,300	119,000	29,700
Black	15-19	1,098,000	90,000	14,300	2,200	500
	20-44	4,984,000	981,800	184,400	34,900	10,000
Total[b]		41,300,000	4,460,600	761,400	161,600	41,800
Pregnant Women in SMSAs[a]						
White	15-19	433,000	39,800	2,200	400	100
	20-44	2,380,000	230,900	42,800	9,500	2,400
Black	15-19	187,000	15,300	2,400	400	100
	20-44	595,000	117,200	22,000	4,200	1,200
Total[b]		3,595,000	403,200	69,400	14,500	3,800

[a] Method of calculating explained in text of Chapter VII.

[b] Totals by addition, not estimated.

These projections for women should be viewed in light of the methodological variables that will contribute to both overestimations and underestimations. The logistic regression analysis accounts for the declines in women's blood lead due to the phasedown of lead in gasoline, but does not account for the reductions of lead in food over this time span. This would result in an overestimation. The original NHANES II survey did not include enough women of "Other Race" to establish statistically reliable prevalences of blood lead levels. Consequently, this total population is excluded from the estimates presented, which are restricted to white and black women. Women of "Other Race" constitute sizable segments of the female populations in SMSAs in the West and Southwest of the country. Finally, the women not residing in SMSAs, about 20%, were omitted entirely from the calculations presented and result in significant underestimations.

Women of childbearing age represent about 45% of the total female population. The prevalence rates for Pb-B levels significant to the impairment of healthy fetal development equate to about 4,460,600 women in the urban population. At any given time, almost 9% are pregnant, and in a given year, about 400,000 pregnancies are at risk for adverse health effects from maternal lead (>10 μg/dl Pb-B). Since pregnant women and this population segment are continuously changing and not readily identifiable, the same quantitative problem recurs until abatement reduces the lead in the environment of these women. In other words, no fixed, identifiable group of individuals has a one-time exposure risk. Over a 10-year period, for example, the cumulative number of individual fetuses at risk will be 10 times that of a single-year tally.

VIII. THE ISSUE OF LOW-LEVEL LEAD SOURCES AND AGGREGATE LEAD EXPOSURE OF U.S. CHILDREN

In Chapter VI, we presented an approximate categorical source ranking for childhood exposure, including some dominant sources or cluster of sources, for example, dusts and soils. This qualitative comparison was reasonable because of the high lead concentrations in the source.

However, the impact of low-level lead sources such as food or water cannot be assessed without simultaneously considering all lead inputs to the body. Exposure estimation strategies based on aggregate lead exposure risk for food intake were employed in Chapter VI. Concern for total input from multiple low-level sources recognizes that lead enters the body from various sources and presents a unified toxicological threat since absorbed lead is toxicologically independent of source. For such multiple low-level intakes and uptakes, we need to examine source contributions as well as changes in Pb-B that occur with changes in low-level sources.

A cumulative exposure approach also requires us to examine various parameters associated with different body organs and functions (e.g., lungs versus the gastrointestinal tract) in the same population group or among various groups (e.g., children versus adults). Details of the metabolic factors appear in Chapter III. The effect of a smaller amount of lead deposited in a body compartment that releases a large percentage of it into the bloodstream may be more severe than a larger amount lodged in a body repository that better retains it.

The issue of aggregate exposure to low-level lead sources has both scientific and regulatory policy aspects. With respect to policy, these aspects include the degree of exposure remediation possible for various lead sources. For example, a blood-lead level of 25 µg/dl can be considered as one index of toxicity. Let us assume that Source A contributes the equivalent of 20 µg/dl, or 80% of this Pb-B, whereas Source B contributes 5 µg/dl or 20%. In theory, removing Source A can lower the Pb-B level by the greatest amount. If this is not possible, but lead reduction in Source B is achievable, then reductions in

Source B are still useful. One can lower the Pb-B level from 25 to 20 μg/dl, which is still below the selected Pb-B toxicity index.

Several approaches to the problem of the aggregate impact from various sources of low lead concentrations need to be examined. National or regional surveys of blood-lead levels would, of course, reflect the total integrated amount of lead absorbed from all sources across national or regional population groups; such surveys could set baselines for comparing changes. Methods for tracing specific sources of blood lead need to be further explored, and the impact of lead distribution in a given source low in lead need to be further evaluated. Quantitative metabolic models of lead intake, uptake, and systemic distribution that permit the factoring of all inputs to Pb-B levels beyond what is now available need further development.

A. NATIONAL/REGIONAL SURVEYS OF BLOOD LEAD LEVELS: BASELINE LEVELS AND SOURCE-RELATED CHANGES IN SURVEY BASELINES

In this section, the impact of regulatory actions on both lead sources and blood lead levels are examined. National survey-based approaches that permit simultaneous examinations of lead source and blood lead trends, as lead sources are regulated, are very useful. These approaches are, however, expensive and complicated.

The best example of this approach, and one that has figured heavily in policy with regulatory aspects of the U.S. lead problem, is the NHANES II national Pb-B survey. The observed declines in national blood lead levels during the survey have been very highly correlated over time with declining use of lead in gasoline.

Figure VIII-1 shows composite plots of Pb-B declines over the course of the NHANES II survey, 1976-1980, and the declines in consumption of leaded gasoline. These declines included both annual and seasonal changes (Annest et al., 1983). The overall level of blood lead declined by 37%. The change was distributed rather uniformly across age and sociodemographic groups and was caused by a pervasive, host-independent source. This is discussed in detail in EPA documents (U.S. EPA, 1985, 1986a).

As a consequence of the quantitative relationships between the rate of decline for leaded gasoline use and Pb-B levels in children (see Chapters V and VI), reductions in the number of children who had some toxicity risk due to

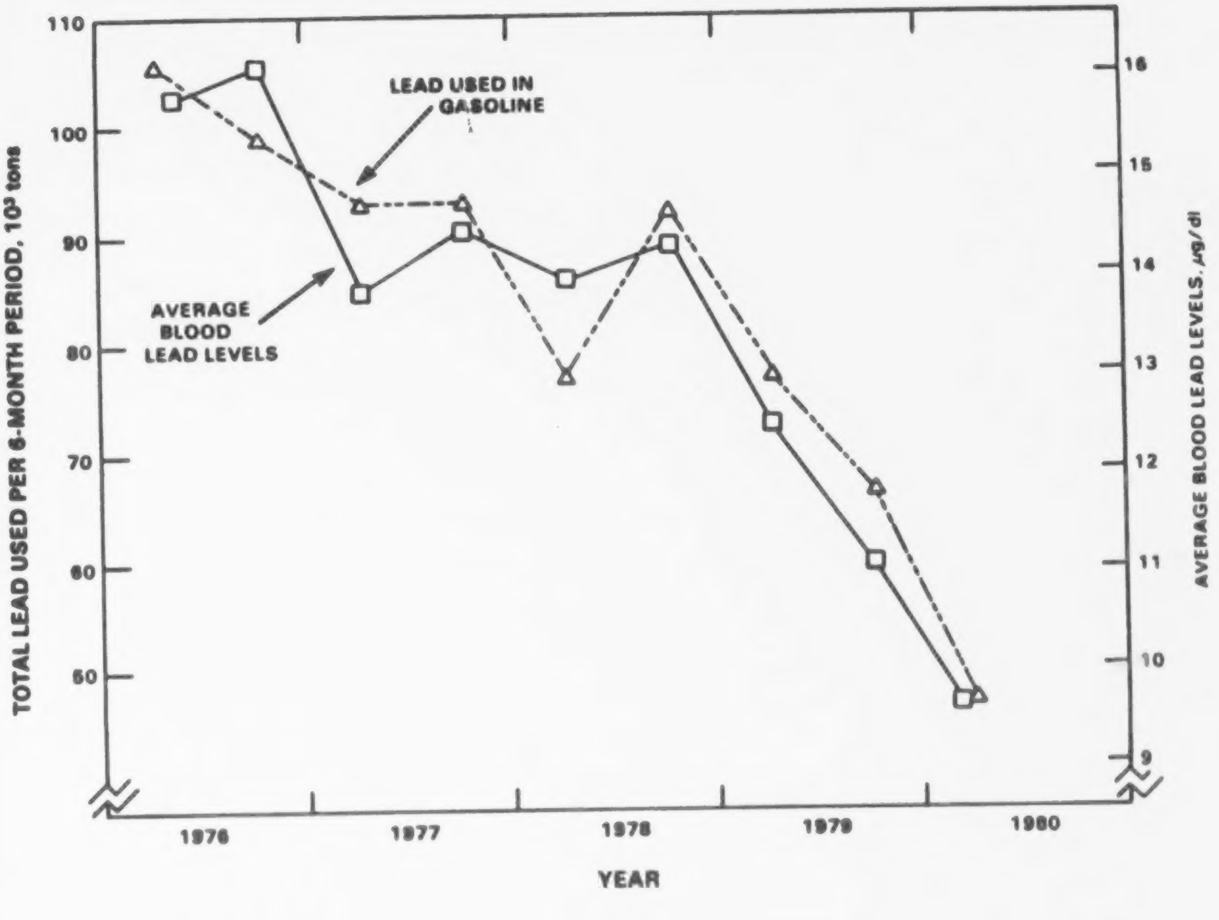


Figure VIII-1. Parallel decreases in blood lead values observed in the NHANES II Study and amounts of lead used in gasoline during 1976-1980.

Source: Annest et al. (1983).

leaded gasoline consumption could be projected. This approach did not require any prior knowledge about the significance of gasoline lead in a steady-state lead-exposed population; one simply examined the results when the relative input of gasoline lead was reduced. Post hoc judgments about the original impact of this particular source can then be made.

B. USE OF SOURCE-SPECIFIC TRACING METHODS

Various geochemical and biochemical principles can be used to develop what are called tracing methods. For instance, atomic lead is composed of various stable isotopes, usually in a fixed ratio, particularly 204: 206: 207: 208. Occasionally, there are isotopic ratios that differ from those in the mainstream of lead-bearing pathways. In that case, one can theoretically "orchestrate" an exposure situation where such an isotopic ratio "tracer" can be used to measure the impact of a specific lead source by using it to examine changes in body lead isotope ratios when the lead first enters the body; we can do this by looking at isotope data in Pb-B. Simple mathematical equations then depict the total body lead burden that arises from the source being traced.

In the Isotopic Lead Experiment (ILE) performed in the Piedmont and the city of Turin in Italy, lead isotopes with an isotope ratio quite different from that in local food and water were used in leaded gasoline, through the cooperation of the lead additive manufacturer and the oil companies (Fachetti and Geiss, 1982; Fachetti, 1985). The lead isotope ratios in ambient air and blood lead levels in communities away from Turin and within Turin were examined over the time that the special mixture in leaded gasoline was used in this area.

Such techniques require a knowledge of the toxicokinetic behavior of lead in human populations. As noted earlier, any portion of this "ratio tag" entering survey individuals and moving to bone for subsequent release (a sizable fraction, as noted in Chapter III), will not appear in the estimates. The bone lead reservoir, where large amounts of lead are stored, contains so much "old ratio lead" that the new ratio lead entering bone cannot survive statistically when it is resorbed into the bloodstream as fully randomized atoms. Therefore, it is lost to any toxicokinetic accounting.

Table VIII-1 shows the estimated lead fraction from leaded gasoline and the portion of this airborne source in blood lead. During the study period, leaded gasoline in Turin was estimated to contribute 87% to ambient air lead and a minimum of about 22% to blood lead; this latter fraction is dependent, of course, on the total Pb-B level. In Turin, the values were in the 20 to 29 µg/dl range. In a population with a similar level of leaded gasoline use, but with a total Pb-B of 10 µg/dl, this contribution would be 47% of total Pb-B. Hence, about 22% would probably be a lower boundary for values in communities with lower total blood lead levels. The fractional value of about 22% is a minimal percentage estimate on metabolic grounds, as noted above. Also, note that these values are for adults; comparable information for children is presently not available.

TABLE VIII-1. ESTIMATED CONTRIBUTIONS OF LEADED GASOLINE COMBUSTION TO BLOOD LEAD BY VARIOUS PATHWAYS[a]

Location	Mean Pb-B (µg/dl)	Pb-B from gasoline (%)	Inhaled fraction
Turin	21.8	4.7 (21.4)	0.6
<25 km	25.1	2.9 (11.4)	0.2
≥25 km	31.8	3.2 (10.1)	0.1

[a] Adapted from U.S. EPA tabulations (U.S. EPA, 1986a) with Fachetti (1985) update.

C. THE USE OF SOURCE-BASED DISTRIBUTIONS OF LEAD INTAKE AND SOURCE-BLOOD LEAD RELATIONSHIPS IN ASSESSING AGGREGATE INTAKE AND POPULATION RISKS

The methods discussed in the previous two sections dealt with the assessment of source-specific inputs into blood lead and will, therefore, be most useful when change occurs only in a specific source or when ascertaining the relative input of one source into total Pb-B levels. To factor more than one changing source into the inputs to Pb-B levels is complicated.

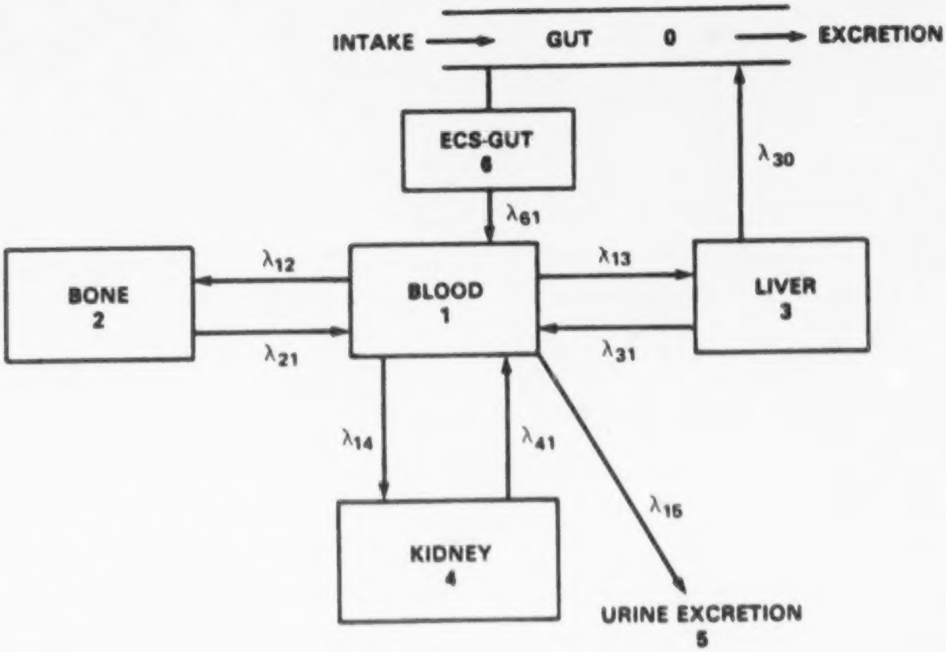
With multiple, low-level lead sources, one can attempt to measure the lead content of each source and use an empirically derived mathematical relationship to relate source lead to some specific Pb-B level in exposed individuals. This is usually done with source-specific regression equations. Being derived from experimental data, these equations are most relevant to the actual design and technical parameters of that specific study. One can extend this approach, with combining of data sets, as the basis of some systematic biokinetic modeling techniques as indicated in the next section.

D. BIOKINETIC MODELS OF THE IMPACT OF LEAD SOURCES ON BLOOD LEAD

In the area of lead toxicokinetics and toxicology, there have been numerous attempts to derive precise models of how external lead relates to body burden and various biological indicators of that burden (e.g., blood lead, see U.S. EPA, 1986a, for a detailed discussion). Such modeling attempts have differed in their complexity and applicability across a range of exposures. For relatively moderate lead exposure, the model of Kneip et al. (1983) accounts for transfer and absorption coefficients relevant to the developing child and involves manageable first-order kinetic solutions. This multicompartment model, developed from (infant) nonhuman primate metabolism of ingested lead, is depicted in Figure VIII-2.

One advantage of such quantitative estimation models is that if knowledge of external media lead content is available, data for such lead levels can be inserted into the computations used to obtain Pb-B levels. This practice involves use of computers and specialized computer programs. Using models to examine Pb-B levels is useful in obtaining at least general assessments of a population's risk for lead exposure/toxicity when field studies, such as the screening for elevated Pb-B levels, are not or cannot be done.

The U.S EPA Office of Air Quality Planning and Standards, (OAQPS) has applied the Kneip et al. model to young children to depict short-term exposures (U.S. EPA, 1986c; Harley and Kneip, 1985; Johnson and Paul, 1986). This U.S. EPA model is defined by its name: Integrated Lead Uptake/Biokinetic Model. The model assesses relative impacts of lead from different sources by integrating the data for all absorbed lead via different media and different absorption rates and by allowing the grand total of absorbed lead to be metabolically integrated to yield levels of lead in the bloodstream and other parts of the body. Since blood is also the biological monitor, it reasonably expresses what is going on internally. In fact, a major use of modeling is to provide the theoretical underpinning for choosing among biological monitors. This topic is discussed in detail elsewhere (Mushak, 1986; Elinder et al., 1987).


INTAKE
GUT
0
EXCRETION
ECS-GUT
6
λ_{30}
λ_{61}
λ_{12}
BONE
2
BLOOD
1
λ_{13}
LIVER
3
λ_{21}
λ_{31}
λ_{14}
λ_{41}
KIDNEY
4
λ_{15}
URINE EXCRETION
5

λ_{12} = 0.34 (INFANT) = 0.11 (JUVENILE)
λ_{21} = 1.73×10^{-3}
λ_{13} = 0.10
λ_{31} = 0.03
λ_{14} = 0.03
λ_{41} = 0.07
λ_{15} = 0.08
λ_{30} = 0.01
λ_{61} = 0.23

Figure VIII-2. Schematic model of lead metabolism in infant baboons, with compartmental transfer coefficients.

Source: Kneip et al. (1983).

Table VIII-2 presents the varying intake/uptake (absorption) estimates for 2-year-old children under three exposure scenarios and the resulting different Pb-B levels. The data utilized are currently available for air quality, soil, dust, and dietary lead levels among such children. The three scenarios are: (1) urban non-point source area; (2) point source impact area; and (3) urban area with leaded paint.

Household dust from chalking was used as the source of leaded paint exposure in the table's scenario, which markedly underestimates overall leaded paint input, since the ingestion of leaded paint (for example, eating flaking paint or gnawing lead-painted woodwork by children) is not included. Such exposure would be difficult to quantify in any case. This scenario also does not include exterior paint weathering.

Direct and indirect (fallout) impacts of airborne lead emissions from nearby sources are shown in the table. Distance and wind direction from point sources cause sharp differences in lead exposure. The situation illustrated in the table only represents a portion of the sensitive population who may be exposed around a given source. When comparing urban children exposed to lead from paint (third column; all other exposure sources being held constant) with urban children not exposed to leaded paint (first column), the first group has upper and lower Pb-B bounds approximately 3.3-fold higher than the latter.

In lead toxicity assessment, distribution of Pb-B levels within a risk population is of special concern, perhaps even more than the actual mean values. The EPA model produces geometric mean blood levels, and the OAQPS draft report indicates how to derive distribution estimates from these model-based means, given geometric standard deviations (GSDs) for the typical log-normal distributions of Pb-B values.

To be useful, models must be validated with empirical information. The validation exercise done to test the predictive accuracy of the Uptake/Biokinetic Model drew upon results of a 1983 survey around the primary lead smelter in East Helena, MT (CDC, 1986a). The survey was conducted jointly by CDC, EPA, and the Montana Department of Health and Environmental Services. Study area parameters, at various distances from the smelter, were examined including dust, soil, and blood lead concentrations of children up to 5 years old. Available aerometry and emissions data were used to estimate airborne lead exposure at different locations in the area.

TABLE VIII-2. ILLUSTRATIVE MODELING BALANCE SCHEMES FOR AVERAGE LEAD INTAKE AND UPTAKE IN 2-YEAR-OLD CHILDREN UNDER THREE SCENARIOS[a,b]

	Parameter	Urban Non-Point Source Area	Point Source Impacted Area	Urban Area With Leaded Paint
1.	Outdoor air lead (μg/m^3)	0.25	1.0	0.25
2.	Indoor air lead (μg/m^3)	0.08-0.2	0.3	0.08-0.2
3.	Time spent outdoors (hours/day)	2-4	2-4	2-4
4.	Time weighted average (μg/m^3)	0.09-0.21	0.36-0.42	0.09-0.21
5.	Volume of air respired (m^3/day)	4-5	4-5	4-5
6.	Lead intake from air (μg/m^3)	0.4-1.1	1.4-2.1	0.4-1.1
7.	% Deposition/absorption in lungs	25-45	42	25-45
8.	Total lead uptake from lungs (μg/day)	0.1-0.5	0.6-0.9	0.1-0.5
9.	Dietary lead consumption (μg/day)			
	a) from solder or other metals	6.1	6.1	6.1
	b) atmospheric lead	4.5	4.5	4.5
	c) natural lead, indirect atmospheric undetermined sources	4.4	4.4	4.4
10.	% Absorption in gut	30-40	30-40	30-40
11.	Dietary lead uptake (μg/day)	4.5-6.0	4.5-6.0	4.5-6.0
12.	Outdoor surface soil/dust lead (μg/g)	55-200	400-975	55-200
13.	Indoor dust lead (μg/g)	85-225	785-1350	2000
14.	Time weighted average (μg/g)	80-217	721-1225	1352-1700
15.	Amount of dirt ingested (g/day)	0.085-0.13	0.085-0.13	0.085-0.13
16.	Lead intake from dirt (μg/day)	6.8-28.2	61.3-159.3	114.9-221.0
17.	% Dirt lead absorption in gut	40	20	20
18	Lead uptake from dirt (μg/day)	2.7-11.3	12.3-31.9	22.9-44.2
19.	Total lead uptake from lung and gut (μg/day)	7.3-17.8	17.4-38.7	27.6-50.7
20.	Average blood lead (μg/dl)	3-7	6-15	11-20

[a] Range of average blood lead levels at age 2 reflect exposures from birth. Uptake estimates for first 2 years of life are calculated and incorporated but not displayed here. Differences in earlier years are due to: (1) higher urban air, soil, and dust lead levels in early 1980s; (2) lower uptake levels during the first year from inadvertent dirt consumption when hand to mouth activity is relatively low; and (3) higher dietary lead consumption rates in early 1980s before reductions of lead in canned foods that have continued over the years. Age-specific exposure parameters used for first 2 years of life are as follows:

Age:	0-1 year	1-2 years
Time spent outdoors (hours/day)	1-2	1-3
Volume air respired (m^3/day)	2-3	3-5
Dietary lead consumption (μg/day)	11.5	12.2
% absorption in gut	42-53	42-53
Amount of dirt ingested (g/day)	0-0.85	0.085-0.13

[b] Calculations as provided by the EPA Office of Air Quality Planning and Standards, October, 1987.

The model described here yields geometric mean and median Pb-B levels in children having multimedia lead exposure. The model's predictive power increases with the level of certainty about all source variability. OAQPS has used exploratory analyses to examine prediction/observation comparison runs for two cases (Run 1, Run 2) where individual dust/soil measures inside and outside of homes were included. In the exercise, a third run also generated prediction curves when a generalized measure of soil/dust lead was included. Figure VIII-3 depicts predicted versus measured Pb-B levels in children 1 to 5 years old living within 1 mile of the smelter operation. Measured soil and dust levels were used as analysis inputs to obtain Figure VIII-3. Plots based only on estimated soil and dust lead levels show similar patterns, although Pb-B levels closest to the smelter are slightly underestimated. OAQPS is continuing to examine these data.

Table VIII-3 presents predicted versus measured Pb-B level in the two areas (Area 1, ≤1 mile; Area 2, 1 to 2.25 miles) as a function of measured versus estimated soil/dust lead levels. Overall, there was reasonably good agreement between the two values.

In response to ATSDR's request for testing the model in other exposure cases, OAQPS compared the model estimates with earlier empirical information for Omaha, NE, and for Silver Valley, ID, two areas in which extensive studies have been done on children affected by lead operations.

Table VIII-4 presents predicted versus measured/reported data for the Omaha investigations (see Angle et al., 1984, and references cited therein). Note that although the predicted values and measured values are similar in the suburban site, the model underestimates the levels in the mixed commercial/residential area. One problem with the Omaha data is the nonspecificity of the soil/dust measures, measures that are present and precisely established in the East Helena survey used to validate the model. In Omaha, furthermore, socioeconomic and other factors may have increased blood lead levels beyond those predicted by the model. For example, much higher historical airborne lead exposures than during the study year were noted.

Table VIII-5 shows predicted versus measured Pb-B values in the Silver Valley, ID, area, where much childhood exposure information was gathered in the 1970s (Yankel et al., 1977). Note that agreement increases with distance from smelter (Area I is closest, Area VII is furthest), as would be expected, given that the Pb-B versus lead-intake relationship is based on linear kinetics that

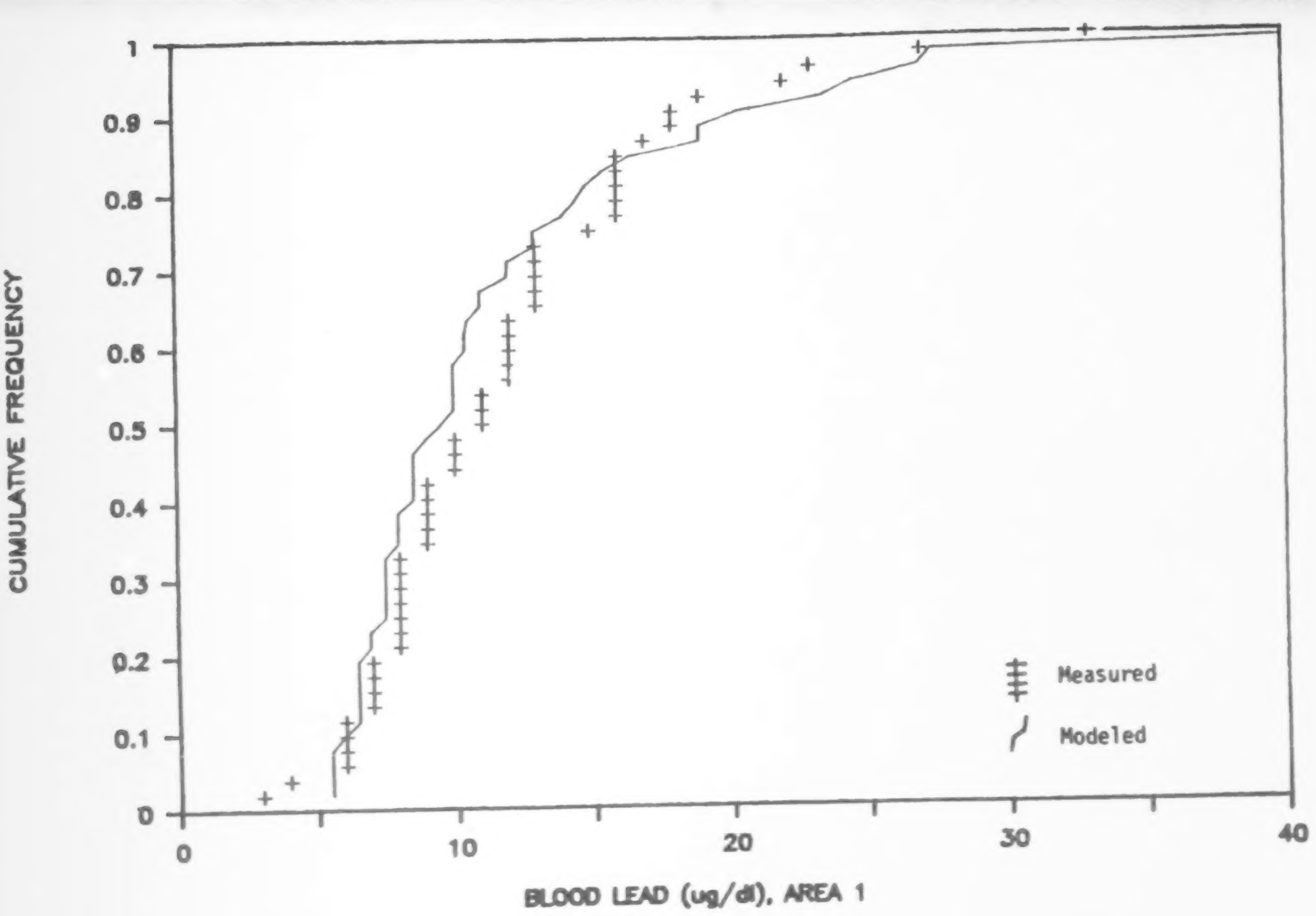


Figure VIII-3. Comparison of distribution of measured blood lead levels in children, 1 to 5 years of age, living within 1 mile of E. Helena lead smelter vs. levels predicted by uptake/biokinetic model. Measured soil and dust levels were included in estimating uptake levels.

Source: Data provided by OAQPS to ATSDR, January 1987.

TABLE VIII-3. COMPARISON OF INTEGRATED LEAD UPTAKE/BIOKINETIC MODEL PREDICTIONS TO 1983 MEASUREMENTS IN EAST HELENA, MT[a,b]

Model Run #	Child Population[c]	Airborne Lead	Dust/Soil Lead	Predicted vs. Measured Average Blood Lead
1	28 living in Area 1 around single air monitor with valid data	Measured at monitor approx. 1/4 mile from smelter	Measured in- and outside individual homes	16.0 vs. 16.5 µg/dl
2	89 Area 1 and 210 Area 2	Contribution from smelter emission estimated by dispersion model; (Runs 2 & 3) local background lead levels (e.g., auto emissions, fugitive dusts) estimated from 1982 source apportionment study	Measured in- and outside individual homes	Area 1: 11.5 vs. 11.8 µg/dl Area 2: 8.9 vs. 8.7 µg/dl
3	89 Area 1 and 210 Area 2		Estimated from generalized air: soil/dust lead relationships from available data in literature	Area 1: 10.3 vs. 11.8 µg/dl[d] Area 2: 9.1 vs. 8.7 µg/dl

[a] Dietary lead exposure estimated for all model runs based on year-specific analyses of 1980s FDA food lead concentration data and food consumption data from Pennington (1983; see U.S. EPA, 1986a), and USDA Nationwide Food Consumption Survey, 1977-1978 (see U.S. EPA, 1986a).

[b] Source: OAQPS/EPA analyses: October, 1987.

[c] Areas as defined in the text.

[d] Preliminary estimates; analysis using generalized soil/dust lead estimates undergoing reexamination.

TABLE VIII-4. CHILDREN'S BLOOD LEAD LEVELS MEASURED IN OMAHA, NE, 1971-1977 vs. INTEGRATED UPTAKE/BIOKINETIC MODEL PREDICTIONS[a,b] AND INCLUDING SOURCE LEVELS

Parameter	Mixed Site	Suburban Site
Observed mean Pb-B (μg/dl) 1-5 year olds	25.6	14.6
Predicted mean[c] Pb-B (μg/dl) 2 year olds	18.5	14.8
Average air lead (μg/m^3)	0.26	0.37
Average soil lead (ppm)	213	110
Average house dust lead (ppm)	653	567

[a] Estimated dietary lead uptake (micrograms/day) for both sites during early 1970s; based on FDA food lead and food consumption data analyzed in U.S. EPA (1986a):

Age (years)	Lead in Food Consumption
0-1	16.3 - 20.6
0-1	16.3 - 20.6
1-2	20.2 - 25.6
2-3	16.6 - 22.1

[b] Source: OAQPS/EPA analyses, October, 1987.

[c] Predicted mean Pb-B represents average of lower- and upper-bound means estimated by model.

cannot be extrapolated reliably to the very high exposure situation as seen in Areas I and II in Silver Valley.

E. SUMMARY

To assist the general reader in a better understanding, this chapter wrestles with the following problem. Assume an individual is exposed to five lead sources simultaneously and each source is low enough in lead that it only contributes 6 μg/dl to that individual's Pb-B level. If each source were judged in isolation, only a low to moderate toxicity risk might be perceived for that individual. Unfortunately, the body integrates these individual contributions (it adds up all five Pb-B increments in this case) into a single toxicological lead burden for health effects and this individual will therefore

TABLE VIII-5. CHILDREN'S BLOOD-LEAD LEVELS MEASURED IN SILVER VALLEY, ID, 1974-1975 vs. INTEGRATED UPTAKE/BIOKINETIC MODEL PREDICTIONS[a,b]

Parameter	Area[e]						
	I	II	III	IV	V	VI	VII
Observed Pb-B (μg/dl) 2-year olds	72	51	36	35	35	25	35
Predicted mean Pb-B[c] (μg/dl) 2-year olds	87.8	73.5	26.8	32.0	33.9	20.6	34.2
Average air lead ($\mu g/m^3$)[d]	16.8	14.2	6.6	3.0	0.7	0.5	0.5
Average soil lead (ppm)	1,470	3,300	1,250	1,400	2,300	337	700
Average house dust lead (ppm)	11,700	10,300	2,400	3,300	3,400	1,800	3,900

[a] Estimated dietary lead uptake (micrograms/deciliter) for all sites during early 1970s based on FDA food lead and food consumption data analyzed in U.S. EPA (1986a):

Age (years)	Lead in Food Consumption
0-1	16.3 - 20.6
1-2	20.2 - 25.6
2-3	16.6 - 22.1

[b] Source: OAQPS/EPA analysis, October, 1987.

[c] Predicted mean Pb-B represents average of lower- and upper-bound means estimated by model.

[d] Airborne lead levels provided by Idaho Department of Health and Welfare.

[e] Area refers to distance from smelter, with Area I being closest.

have a measurable Pb-B of 30 μg/dl. For a young child or a pregnant woman (fetal exposure), a Pb-B of 30 μg/dl poses an unacceptable toxicity risk and requires immediate reduction.

Following comprehension of the above problem's impact, the question of how best to manage such low-level sources remains. When there is one large lead source or a dominant source associated with severe lead intoxication, the exposure abatement remedies are more apparent and justifiable. With the above example, which of the five low-level sources first should receive attention?

In reality, we qualitatively recognize that a number of low-level lead sources are operating in the human environment. However, we often do not know their quantitative impacts on Pb-B levels unless we track one source environmentally and/or metabolically or use an integrated approach in which we measure all levels and estimate Pb-B contributions from each of these sources. In this chapter, both approaches were described and their relative value and limitation discussed.

Single-source changes can be examined by careful trend analysis of Pb-B changes versus trends in the source contributing to Pb-B. The observed high correlation between declines in leaded gasoline use and declines in nationwide U.S. Pb-B levels illustrates this.

A second, conceptually distinct strategy is to trace source-specific lead through the environment and after it enters the human body. An example is the Turin, Italy, gasoline lead experiment studying a distinct elemental isotope composition used as a tracer.

The net result of both of these approaches was the determination that gasoline lead as a nationwide exposure source was contributing at least 25 to 50% of body lead in all members of the population, and gasoline lead reduction in response to regulatory initiatives will, cumulatively, reduce or avert unacceptable levels of Pb-B in millions of young children.

The two approaches presuppose that only one lead source, or one main lead source, is being changed in some fashion, e.g., total level of lead or its isotopic composition. When all or most lead sources with which individuals have contact are changing, assessment of these changes becomes more complicated.

One strategy being pursued by various researchers and agencies is the use of a cumulative metabolic model. In such idealized modeling approaches, as presently defined, levels in each exposure medium are measured and the data employed to calculate a total Pb-B level, using appropriate mathematics and computer programs. If Pb-B levels are estimated as high, then scenarios that reduce Pb-B in various media can be examined. At present, such modeling approaches for moderate overall exposure appear encouraging in the case of children. Modeling of aggregate exposure risk for the fetus (due to elevated body lead in pregnant women) is potentially more complex. For such modeling approaches to be useful, they have to account for the Pb-B distributions arising for a set of individuals having identical exposure. Approaches being developed now attempt to do this. A related approach uses distributions of lead levels from a given source, if available, to define risk populations by source intake and uptake above some cumulative frequency distribution of Pb-B levels.

As the index of lead body burden, i.e., blood lead level, is revised further downward in terms of perceived unacceptable toxicity risk, the problem of cumulative impacts of only low-lead sources becomes magnified. For example, when we say that a Pb-B level of about 10 μg/dl is associated with onset of toxicity risk in the human fetus, we can return to our earlier example of five low-lead sources and substitute contributions of only 2 μg/dl each to yield a total that is equal to the criterion Pb-B level of 10 μg/dl. If we accept 10 μg/dl in pregnant women to be the maximum tolerated, for valid reasons given in Chapter IV, then the average Pb-B in the population necessary to avoid this must be considerably lower than 10 μg/dl. The same concerns apply for young children. The implication of this is that control options increasingly will center on producing reductions in ever smaller Pb-B increments.

IX. METHODS AND ALTERNATIVES FOR REDUCING ENVIRONMENTAL LEAD EXPOSURE FOR YOUNG CHILDREN AND RELATED RISK GROUPS

Section 118(f) of SARA directs that ATSDR examine methods and alternatives for reducing environmental lead exposure in young children. This topic encompasses many environmental and social issues and only a limited number of them can be discussed in this report.

The collective sense of the earlier chapters is that a significant problem remains with certain source-specific lead exposures and toxicity among young children and other U.S. risk groups. With other sources, specific measures with large consequences for exposure control have been put in place in the United States. These are helping to reduce some of the original levels of exposure and toxicity in identifiable segments of the risk populations.

Questions surround the issue of adequate environmental lead reduction. Is it simply bringing exposed populations below some Pb-B value associated with some adverse health risk? Alternatively, is it reducing population exposure to a level that also allows some modest margin of safety? This safety margin is desirable for obvious reasons, not the least of which is minimizing toxicity risk. Any likelihood that future information will cause further downward revisions in acceptable levels of Pb-B is a second reason. One can only surmise what the positive public health benefits of safety margins might have been earlier, when toxicity was deemed to be at Pb-B levels of 60 to 80 μg/dl. An additional point is the unique position of lead in terms of safety margins among human toxicants. There is virtually no margin between observed levels measured in the population and the level at which effects appear.

The topic of exposure prevention methods and alternatives is best addressed in two parts: primary prevention and secondary prevention. It is a common practice to classify prevention strategies into primary, secondary, and tertiary approaches. However, for our purposes, tertiary strategies are more clearly presented under parts of secondary prevention. The components of each type of prevention method are depicted in Table IX-1.

TABLE IX-1. CATEGORICAL TABULATION OF THE COMPONENTS OF PRIMARY AND SECONDARY PREVENTION OF LEAD EXPOSURE IN CHILDREN AND RELATED U.S. RISK GROUPS

Type of Prevention Method	Components of the Method
I. Primary	
A. Environmental	1. Lead in paint 2. Lead in ambient air (a) Leaded gasoline combustion (b) Point source emissions 3. Lead in dusts/soils 4. Lead in drinking water 5. Lead in foods
B. Environmental/Biological	Source controls augmented by community-nutrition interventions, i.e., nutritional supplementations, for: (a) Calcium and (b) Iron
II. Secondary	
A. Environmental	1. Case finding 2. Screening programs 3. Environmental follow-up 4. Event-specific exposure abatement
B. Environmental/Biological	Nutritional assessment and follow-up on *ad hoc* identification basis
C. Extra-environmental	Legal actions and strictures

Primary and secondary types of prevention of lead exposure and public health risk draw upon classical distinctions made between the two in community medicine (see, e.g., Elinder et al., 1987). Primary prevention strategies span from the very beginning of the commercial existence of a potential human toxicant and extend to controls on the amount of the toxicant permitted to enter both human exposure pathways and the mainstream of economic activity; for example, discontinued use of leaded paint and removal of old paint. Secondary methods of prevention are technically reactive in nature, i.e., a cluster of responses to existing and identified problems. These include preventing the flaking of old paint, maintaining a high level of hygiene, preventing access of children to paint flakes, and minimizing contact with lead in dust. Also, screening for actual lead exposure is considered secondary prevention.

In no small measure, past and current problems with lead as a health risk are traceable to failures in primary prevention mechanisms. For example, adequate safety assessments for leaded paints and leaded gasoline, as we now define them, were not originally applied to these sources. According to both Rosner and Markowitz (1985) and Hamilton et al. (1925), reviewers examining the use of leaded gasoline at either end of a 60-year span, the introduction of tetraethyl lead as a gasoline antiknock additive was permitted in the absence of any credible public health risk assessment. Present U.S. regulatory practices would not permit very many uses of lead if it were in new products, given either the earlier (1920s, e.g., Hamilton et al., 1925) or current toxicology literature on results from experimental test animals and production worker exposure.

As Farfel (1985) has noted, we can further define two approaches under the rubric of primary prevention that have a bearing on lead exposure: passive measures or community-level exposure prevention, and active measures, which require the individuals' participation and changes in their behavior. Active approaches are more difficult to accomplish given both the pervasive nature of lead exposure and requirement for adherence to a preventive behavior pattern. Behaviors of people, however, can be positively changed with intensive public education, as demonstrated in the case of cigarette smoking.

One can also dichotomize primary and secondary prevention strategies along lines of environmental exposure exclusively or environmental control in tandem with biological reduction of in vivo exposure and toxicity risk. This approach combines environmental lead control and biological factors to achieve protection greater than that obtainable from environmental abatement alone. For example, nutritional factors in young children can reduce to some degree lead absorption from the gastrointestinal tract, e.g., adequate iron, calcium, and phosphorus. It is crucial to note that optimizing nutrition is no substitute for environmental control action.

In addition to specific primary and secondary measures for preventing lead exposure, other actions can have the net effect of being prevention measures without being defined as such. For example, the national urban renewal programs of the 1960s and 1970s had the net effect of removing large numbers of inner-city, lead-painted housing units and associated dust/soil surfaces. Whatever the larger societal merits of such measures, at least some degree of lead exposure was removed for inner-city children. Further benefits occurred wherever the land-use changed from residential use and badly contaminated soils and dust areas were made unavailable to children or paved over. Such measures may, on balance, abate risk for a given group, but they pose the ultimate question of where to deposit or dump the lead-bearing material.

A. PRIMARY PREVENTION MEASURES FOR LEAD EXPOSURE

This subsection is divided into environmental measures aimed at preventing exposure, and environmental/biological measures aimed at minimizing the effects.

1. Primary Prevention Using Environmental Measures

This subsection is organized by environmental source category. Of particular interest are data showing how refractory each source may be to collective exposure abatement and control.

Primary prevention as applied to the lead problem has actually been a hybrid of classically defined primary prevention measures and post hoc decisions for exposure reduction that resemble secondary prevention approaches.

a. Lead in Paint

Leaded paint was introduced in the United States with little consideration of any future environmental health concerns. National and other actions to control leaded-paint exposure were only instituted after lead poisoning problems had been recognized (see the history of lead in the United States in Chapter II). Discussion of these levels of action are divided into Federal and non-Federal controls.

Federal Actions in Preventing Paint Lead Exposure in Young Children. Federal actions directed to primary prevention of leaded paint exposure in children concern mainly those taken by the Department of Housing and Urban Development (HUD) and the Consumer Product Safety Commission (CPSC).

The main action of the CPSC relevant to this topic was to mandate reduction of lead in paint to 0.06% lead in 1977. This move primarily affected the rate of further input of leaded paint into the housing stock. The Commission's mandate does not allow addressing the preexisting paint lead burden in U.S. housing stock. Reduction to a level of 0.06% followed an unofficial voluntary restriction by the manufacturers themselves to a 1% lead content in the late 1950s. However, between the 1950s and 1977, paint stocks in excess of this lead level continued to be produced. This level of 1% (as dry solid) still amounted to 10,000 ppm lead, a level well above that associated with elevated Pb-B levels (see Chapter VI).

In contrast to the role of CPSC, HUD has been primarily concerned with leaded paint already present in public housing or any other housing involved in any type or level of Federal assistance; however, HUD did restrict the use of high-lead levels in paints in housing stock under its jurisdiction. The Lead-Based Paint Poisoning Prevention Act (42 U.S.C. 4801 et seq.) authorized HUD action to prohibit the use of leaded paint in Federal or Federally assisted construction or rehabilitation; relevant HUD regulations were adopted in 1972. A major statutory step forward in HUD's responsibilities was mandated in Section 302 of the Act, added in 1973, which required HUD to set up procedures for leaded paint abatement in existing housing stock. Here also, jurisdiction was limited to Federally connected housing.

In 1973 and again in 1976, HUD acted in two ways under provisions of Section 302: (1) warnings to purchasers and tenants of HUD-associated housing as to "immediate hazard" in housing built before 1950, and (2) prohibiting lead-based paint at a level above 0.5% (prior to the 0.06% level as of June 22, 1977). Recently, HUD has become even more involved as a result of 1983 court action (Ashton vs. Pierce, 716 F.2d 56/D.C. Cir. 1983). This action challenged HUD regulations to include essentially all lead-painted surfaces as an "immediate hazard" rather than just the criteria of conditions associated with deteriorating surfaces and the dwelling in general. HUD has, at present, promulgated three rules that extend considerably its activities in this area: (1) lead paint hazard elimination in public and Indian housing (51 FR 27774: August 1, 1986; effective September 23, 1986); (2) lead paint hazard elimination in FHA single- and multi-family units and Section 8 housing/housing voucher and rehabilitation, FHA single- and multi-family property disposition (foreclosure) programs (52 FR 1876: January 15, 1987; effective date, March 2, 1987); and (3) lead paint hazard elimination in various community-based Federal grant and related programs (52 FR 4870: February 17, 1987; effective date, March 19, 1987).

Collectively, these new actions address virtually the full spectrum of U.S. housing activity in which HUD has some assistance role. However, no Federal action exists to reach directly into fully private sector housing beyond the lead level in paint offered for sale. The Veterans' Administration and the U.S. Department of Agriculture apparently have not addressed the problem.

The actions concerning public and Indian housing include required inspections for defective paint surfaces in units with children less than 7 years old and required inspections for chewable and defective surfaces if a child has an elevated Pb-B level. The test threshold for paint lead in all cases is 1 mg/cm^2 lead. Recognized problems with the lead detectors, pointed out by HUD in all of the Federal Register notices, cause concern about such testing. Only a limited number are available in the country and their accuracy may be questioned. Current usual operator skills require uniform training and protocols for testing them by a central authority to ensure widespread implementation of the new rules. The new action for public housing requires hazard abatement, i.e., leaded paint removal, when a child is identified with an elevated Pb-B in the dwelling, in common areas, or in public child care facilities within control of public housing. The degree of abatement is linked to such factors as the Pb-B level, local and state practices, and feasibility. While abatement methodologies are not specified, they require that the hazard be "thoroughly removed or covered." Public housing authorities may request Federal funds to carry out abatement if other support is not available.

New HUD activity on the leaded paint hazard in FHA and related housing supported by Federal assistance has a 1973 construction cutoff, i.e., housing built in this year and earlier is covered under the action. Inspection for defective surfaces, as with the public/Indian housing action, does not require X-ray fluorescence analysis, but the chewable, protruding surfaces do. The many components to this rulemaking and the bulk of detail are beyond the scope of this report. They are published in the Federal Register notice (52 FR 1876: January 15, 1987). However, note that testing and abatement actions for FHA-assisted housing are triggered by change in ownership status and continuation of Federal mortgage insurance. Presumably, if a leaded paint-contaminated unit remains in its present ownership status or is bought through non-Federal financing, then the particular requirements do not apply.

With respect to the various community grant-based, Federally assisted programs, most elements of the new rules operationally overlap with the other two sets of actions described above. In brief, this action requires that Community Development Block Grant, Urban Development Action Grant, Secretary's Fund, Section 312 Rehabilitation Loan, Rental Rehabilitation and Urban Homesteading Program applicants must carry out lead paint analysis and abatement steps in order to receive funds within the programs. Both this cluster of HUD community grant programs and that involving FHA-related assistance primarily place abatement costs on the private sectors involved in the housing transactions.

While these recent actions suggest a new comprehensive effort to attack the leaded paint hazard, quantifying the likely or estimated impact of the three rulemaking actions is still necessary. Table IX-2 provides estimates of the number of units and associated abatement costs in public housing, at a paint lead removal action level of 1 mg/cm^2 (Wallace, 1986). About 308,000 units are estimated to require abatement across all unit age categories with an aggregate cost of $380.1 million.

Table IX-3 presents the estimated number of units requiring lead abatement for each year, 1987-1991, and the projected cost in these years for FHA single-family units. For all housing ages, 171,300 units are estimated to require abatement for each of the 5 years, and total 856,500 units with a cost of about $2 billion. Single family, FHA-insured units are but one category in this particular HUD action. Miller and Toulmin (1987) have estimated that for 1987-1991, all of these FHA categories will involve an outlay of $2.57 billion. Of these amounts, about 95% will have to be paid by buyers and/or sellers in the private sector.

Municipal and State Actions in Leaded Paint Exposure. In 1951, the City of Baltimore prohibited leaded paint use on interiors of dwelling units and, in 1958, required warning labels on cans of leaded paint already in the market pipeline (Baltimore City Health Department, 1971). By that time, the paint industry had introduced titanium dioxide as a substitute pigment for lead carbonate in paint, but the advantages of coverage and perceived surface freshness of leaded paint (see Chapter II) assured its persistence at some concentration into the 1970s.

Retroactive regulation at any level of jurisdiction, i.e., states or cities, for paint lead already in U.S. housing stock has been infrequent and

TABLE IX-2. ABATEMENT COSTS AND NO. OF UNITS FOR DIFFERENT SITE CATEGORIES AT A LEADED PAINT THRESHOLD OF 1.0 mg/cm^2 IN PUBLIC HOUSING[a]

Construction Year	Family Dwelling Units		Cabinet Surfaces		Housing Project Units		Common Activity Sites	
	No.	Cost ($Millions)	No.	Cost ($Millions)	No.	Cost ($Millions)	No.	Cost ($Millions)
Pre-1950	81,379	86.7	5,399	3.4	11,239	25.7	413	0.7
1950-1959	111,688	108.6	3,609	2.2	16,808	61.0	425	0.5
1960-1972	114,587	62.8	0	0	11,361	28.2	457	0.3
Total	307,654	258.1	9,008	5.6	39,408	114.9	1,295	1.5

[a] Adapted from Wallace, 1986; number of units indicated is 48.9% of the total of 629,004, and total cost = $380.1 million.

TABLE IX-3. ESTIMATED ABATEMENT COSTS AND NO. OF UNITS FOR DIFFERENT SITE CATEGORIES AT A LEAD PAINT THRESHOLD OF 1.0 mg/cm^2 IN SINGLE-FAMILY FHA HOUSING UNITS[a]

Year Built	Year of Abatement-Number of Units				
	1987	1988	1989	1990	1991
1960-1972	20,500	20,500	20,500	20,500	20,500
1950-1959	55,900	55,900	55,900	55,900	55,900
Pre-1950	94,900	94,900	94,900	94,900	94,900
Total No.	171,300	171,300	171,300	171,300	171,300
Total Cost ($ Thousands)	388,400	388,400	388,400	388,400	388,400
Cumulative Cost ($ Thousands)	388,400	776,800	1,165,200	1,553,600	1,942,000

[a] Source: Miller and Toulmin (1987).

variably enforced. In the early 1970s, Philadelphia, PA, had a primary prevention ordinance directed at removing leaded paint up to 5 feet above the floor in any unit with leaded paint. However, the city eventually discarded such prophylactic removal in favor of abatement only after demonstrated toxicity in child residents.

Among the states, Massachusetts banned lead in any unit in which children younger than 6 years of age were living, but the combined effect of organized opposition from real estate interests and limited funding for enforcement resulted in secondary prevention--that is, intervention only after demonstrated instances of toxicity (Needleman, 1980).

The Massachusetts statute typifies primary prevention legislation that has been rendered ineffective, for whatever reason. Summary statistics provided by the Commonwealth of Massachusetts to ATSDR, shown in Table IX-4, permit some observations. Of interest is the activity level of lead removal programs, compared with the number of pre-1940 housing units, that is, lead-painted units with high lead content. The table indicates that the selected cities of the Commonwealth have a total of 450,339 pre-1940, high lead-painted units. Over the period January 1982 to June 1986, only 2260 or 0.5% of these units were subjected to lead abatement. We are not aware of the level, if any, of lead removal carried out under Massachusetts statutory provisions but occurring outside the reported programs.

TABLE IX-4. SUMMARY OF TOTAL PRE-1940 LEAD-PAINTED HOUSING VERSUS DELEADING ACTIVITY IN SELECTED MASSACHUSETTS COMMUNITIES FOR 1982-JUNE 30, 1986[a]

City	Pre-1940 Units[b]	Units Deleaded In: 1982	1983	1984	1985	1986[c]	Total
Boston	179,391	221	175	136	201	152	885
Worcester	43,555	148	100	99	142	67	556
Springfield	36,239	40	41	29	34	2	146
New Bedford	29,536	9	21	16	10	1	57
Fall River	28,502	6	5	2	1	0	14
Somerville	26,806	9	7	1	4	9	30
Lynn	26,006	20	29	21	35	12	117
Lowell	23,356	-	-	-	-	-	153[d]
Lawrence	19,916	-	-	-	-	-	300[d]
Newton	18,516	0	1	1	0	0	2
TOTAL	450,339	453	379	305	427	243	2260

[a] Summary Statistics: Childhood Lead Poisoning Prevention Program, Commonwealth of Massachusetts, as provided by Cosgrove to ATSDR, 12/10/86; communities ranked by number of pre-1940 units

[b] All pre-1940 units are assumed to have leaded paint at significant levels.

[c] To June 30.

[d] Total only supplied.

Statutes such as that of Massachusetts can be employed in concerted action by community groups. In 1981, a tract of high-risk, lead-painted housing in the Jamaica Plain area of Boston was systematically examined, the children were screened for lead toxicity, and then 50% of the suspect housing was treated to remove lead. This was brought about by the joint efforts of the Harvard School of Public Health, which did the community assessment, and the Legal Aid Society, which used the Massachusetts statutory sanctions to force the property owners to comply (Harvard School of Public Health, 1981).

A lingering problem with leaded paint is the disposition of old retail stock that has high lead content. CPSC, for example, cannot take action against salvage, close-out, and bankruptcy sales if stock was manufactured before the June 22, 1977 effective date of the 0.06% standard. Because of this and other reasons, high lead-level paints are still circulating in retail channels. The Connecticut Department of Consumer Protection, for example, has noted that lead-based paint can reach the market in higher amounts than expected (Communication of Department of Consumer Protection, State of Connecticut to Dr. Jane S. Lin-Fu, Department of Health and Human Services, September 17, 1985).

In the Connecticut investigation, some of the lead-based paint found during 1985 on retail shelves was over 22 years old. Furthermore, discount and salvage outlets will buy close-out inventories and keep lead-based paint in the consumer pipeline. Of particular concern is the fact that paint producers are permitted to market a "sludge" paint from new materials plus "residues from vats." If these residues are from lead-containing industrial products, then the ultimate lead level in the sludge paint may exceed the CPSC limit of 0.06%.

b. Lead in Ambient Air: Leaded Gasoline Combustion And Point Source Emissions

EPA has had regulatory authority over the use of lead in gasoline since 1973 (24 CFR 965.705(D)(2)). In 1975, EPA classified lead as a criteria pollutant, a designation reserved for pollutants whose public impact is such that control is required by ambient standards rather than by site-specific emission controls. Several parallel actions were being pursued in 1975 under the aegis of either Section 108 or 109 of the Clean Air Act, as amended, USC 7408 and 7409, which authorized the EPA Administrator to set ambient air standards for lead. In addition, Section 211(c)(1) of the Act authorizes the Administrator to: "control or prohibit the manufacture...or sale of any fuel additive" if its emission products cause or contribute to "air pollution which may be reasonably anticipated to endanger the public health or welfare" or "will impair to a significant degree the performance of any emission control device or system...in general use."

Since the mid-1970s the lead use in gasoline began to decline mainly as a result of the increase of lead-sensitive, emission control-equipped vehicles in the U.S. domestic fleet. This downturn has been reasonably established as a significant factor in Pb-B level changes among U.S. population groups, as determined by national (NHANES II, CDC screening data) and regional observations. The NHANES II data indicated a generalized, cross-population decline in Pb-B levels of 37%, an average drop of about 5.4 μg/dl (see U.S. EPA, 1986a, for a detailed discussion). In 1978, the ambient air lead standard of 1.5 $\mu g/m^3$--a considerable drop from the earlier standard--was promulgated. This standard also provided a means for controlling point-source emissions from smelters and similar operations.

In 1982, EPA promulgated new rules (47 FR 49331 October 29, 1982) that, among other things, reduced the lead content of gasoline to 1.1 grams per liquid gallon. This intermediate action coincided with reports of the adverse health effects of lead on children and adults, which argued for still further action, as did the disturbing rate of "misfueling," whereby leaded gasoline was used in vehicles built to use unleaded gasoline. Further action was taken and, effective January 1, 1986, EPA promulgated the phasedown of lead in gasoline to 0.1 g per liquid gallon.

The decline in gasoline lead that these actions will bring is expected to impact the number of children whose Pb-B levels fall below certain toxicity risk ceilings, including the 1985 CDC action level of 25 µg/dl. Tabulations in Chapter VI show expected sizable declines in the numbers of children with Pb-B levels above 15, 20, and 25 µg/dl.

EPA is also examining its 1978 lead standard of 1.5 $\mu g/m^3$ in ambient air, with a likelihood of reducing it. This will reduce atmospheric inputs, mainly in the proximity of stationary sources. As is also the case with post hoc controls on leaded paint, controls on lead inputs from mobile and stationary emissions mainly abate additional exposure. In both cases, populations will continue to be at risk for exposure from lead-contaminated dust and soil, arising from past air lead fallout and lead paint flaking, weathering, and chalking.

c. Lead in Dusts and Soils

The primary prevention measures for exposure to lead-contaminated dust and soil have been directed at the generators of lead for these sources, that is, paint, leaded gasoline, and stationary source emitters. These measures, again, will primarily reduce or eliminate further inputs from these sources.

At present, limited regulatory action seems specifically directed at controlling lead in dust and soil. Chapter X describes Superfund activity and, in Appendix F, lists sites that are due for cleanups and which also contain lead in soil. Several factors have contributed to this lack of regulation. First, dust and soil traditionally have not been recognized in public health actions or policy as specific, potentially major sources or pathways of childhood lead exposure. These sources are complex and still need quantitative characterization. Second, legal and other societal sanctions that are not enforced allow primary contributors, such as leaded paint to continue to contaminate residential dusts and soils. One impediment to regulatory or legal control of lead in dusts and soils has been the relative paucity of studies showing how specific primary contributors affect given dust and soil contamination levels. Duggan and Inskip (1985) have reviewed dusts and soils versus childhood exposure in detail and their review provides further information.

Recent data indicate that some general mix of inputs or specific generators have certain quantitative relationships to Pb-B levels. Charney et al. (1983) have shown that Pb-B levels can be reduced through indoor dust abatement but only to a certain point. Milar and Mushak (1982) have shown a relationship between "occupational" dust brought home by lead battery plant workers and Pb-B levels in their young children. The recent study by Ryu et al. (1985) shows household contamination via secondary transport from the workplace and lead transfer to infants. Reports of the Cincinnati prospective lead studies, concerned with childhood lead poisoning in this city, have shed considerable light on relationships among pathways for household dust, lead on the hands of children, and socioeconomic factors concerning leaded paint as the likely primary contributors (Bornschein et al., 1985; Clark et al., 1985; 1987; Que Hee et al., 1985). Clark et al. (1987) have shown that dust lead is best correlated with lead on the hands of children and point to dust lead abatement as a key factor in reducing lead hazards in housing.

Field studies are needed to provide evidence that "macro" rather than "micro" control strategies are effective means of lead abatement in areas larger than a single home or several homes. The focus of most studies to date has been specific abatement methods that are employed for individual lead paint-containing units. Mobility of lead in dust and soil prevents simple conclusions about single unit abatement to be extended to a neighborhood or even larger area. Field surveys are also needed to define blood lead-source lead relationships. Past attempts to define soil and dust lead in terms of proportional contributions of paint lead or airborne lead when both primary inputs were operative have been unsuccessful for various reasons.

The 1986 Superfund Act provides for the funding and execution of demonstration projects to address the problem of area-wide soil (and dust) lead in urban tracts. In response, in April 1987, EPA conducted an experts' workshop on the design and scientific conduct of soil lead abatement projects. Methods of environmental and biological monitoring as well as the statistical design of the necessary population surveys were discussed. A workshop report was prepared and lays considerable groundwork for future selection of actual demonstration sites and for this general area of exposure assessment.

Recently, a limited but illustrative draft report of alternatives and issues of soil cleanup was prepared by EPA Region I in Boston, with assistance of the Harvard University School of Public Health (Ciriello and Goldberg, 1987). Given the useful nature of the material, the draft report is presented as Appendix E and is only summarized in this chapter.

The report evaluates five proposed remedial alternatives for lead-contaminated soil in urban residential areas. They consist of: (1) removal and off-site disposal of contaminated top soil, with uncontaminated soil coverage and revegetation; (2) the same approach with on-site disposal; (3) covering contaminated points with low-lead topsoil and then revegetation; (4) removal, decontamination, and on-site placement with revegetation; and (5) rototilling soil and revegetation.

To assess the impacts of the five alternatives, a typical site in Boston was identified for each option and comparisons of costs and results attempted. Steps already proposed or implemented at Superfund or other sites also were described and examined. Conclusions of the report include:

(1) Excavation of lead-contaminated soil and on-site decontamination is too costly and operationally unwieldy.

(2) The effectiveness of soil lead abatement steps such as capping, rototilling, excavation, and on-site disposal are uncertain for soil lead levels of 1,000 ppm or higher. On the other hand, they may work for soil with lead levels below 1,000 ppm.

(3) Excavation of lead-contaminated soil with off-site disposal, augmented with Pb-B-level testing for children in the affected residences, seem best for protection; cost and off-site disposal impacts, however, may be a problem.

Note that these strategies represent a limited effort and options germane to the Boston urban area.

d. Lead in Drinking Water

EPA is required, by the 1974 Safe Drinking Water Act (SDWA), to set drinking water standards with two levels of protection. Of interest here are the primary standards for drinking water, which define contaminant levels in terms of maximum contaminant level (MCL) or treatment requirements. MCLs are limits enforceable by law and are to be set as close as possible to maximum contaminant level goals (MCLGs), which are levels essentially determined by relevant toxicologic and biomedical considerations independent of feasibility.

Recently Congress ordered EPA to tighten the drinking water standards for various substances, including lead. The current MCL for lead is 50 µg/l of water. The proposed standard is stricter, 20 µg/l (U.S. EPA, 1986b). In addition to the pending rule on drinking water lead *per se*, the 1986 SDWA amendments ban the use of lead solder and other lead-containing material in household plumbing when residences are connected to public water supplies. The deadline for implementation of the ban is June 1988. States must enforce the ban or are subject to a loss of Federal grant funds.

EPA's Office of Policy Planning and Evaluation (1986b) has carried out a detailed assessment of lead in drinking water from public water supplies. As noted in Chapter VI, about 20% of the population has tap water lead levels above the proposed MCL of 20 µg/l.

Since EPA is concerned with tap water lead levels as well as lead burdens in processed water leaving treatment facilities, the Agency must specify the "best available technologies" for preventing lead entry into drinking water. Proposed are corrosion controls that consist of treating the potable water with sodium hydroxide and lime to raise its pH and alkalinity and adding orthophosphate to aid development of a protective film inside the pipes. In addition, EPA is considering the removal of lead service connections and goosenecks (connections from the street main to house lines) for inclusion in the "best available technologies."

Corrosive drinking water is quite common to high-density U.S. population areas, and U.S. EPA (1986b) has estimated that about 62 million Americans have such drinking water. The best U.S. case study for primary prevention of exposure to lead in drinking water at the community level is that of Boston. In the 1970s, Boston water authorities, knowing that many of the occupied housing units in the city had lead plumbing and that the city's water was highly corrosive, began efforts to reduce corrosivity. These efforts considerably reduced the amount of lead in tap water (see discussion in U.S. EPA, 1986a). In Chapter VI, we noted that the blood lead levels of Boston children had been related to past elevated tap water lead levels. U.S. EPA (1986b) has estimated that the treatment to reduce corrosivity costs just 25% of the value of the health benefits realized from reduced lead exposure, that is, a benefit-to-cost ratio of 4:1. The broader actions under the proposed EPA regulations are expected to have a wide impact on potential childhood lead exposure.

e. Lead in Food

Lead from food and beverages is encountered by virtually the entire U.S. child population and, as shown in Chapter VI, about 5% of the children have a lead intake high enough to result in Pb-B increases causing risk of health impacts. Consequently, prevention measures that limit lead exposure from food are quite important. Regulating lead contamination in foods has been the responsibility of the U.S. Food and Drug Administration (FDA) for several decades and control dates from the identification of lead-containing pesticide residues on sprayed fruits.

Collectively, FDA actions from the 1970s onward have targeted either control through setting total lead intake goals or efforts directed at known significant sources of lead inputs into foods. In 1979, FDA set a long-term goal of less than 100 µg/day for reducing the daily lead intake from all foods for children 1 to 5 years old (FR 44:(171) 51233-51242, 1979). This is a maximum permissible intake for any child and not a mean intake for all children. To achieve this goal within the shortest feasible time, attention focused on (1) establishing permissible lead residues in evaporated milk and evaporated skim milk; (2) setting maximum levels for lead in canned infant formulas, canned infant fruit and vegetable juices, and glass-packed infant foods; and (3) establishing action levels for other foods. Along with these activities, FDA monitors and enforces controls on food-related materials, for example, leaching from pottery glazes and food utensils.

Lead can enter the food supply during production, processing, or distribution. U.S. EPA (1986a) has pointed out that during these activities, the lead content in food may be increased 2-fold to 12-fold over background levels. Processing is the major pathway for contamination--especially lead leached from lead-soldered cans. Since World War II, the ratio of lead to tin in this soldering material has remained at 98:2.

The percentage of food cans that are lead-soldered continues to decline. Table IX-5 shows the percentages from 1979 through the first quarter of 1986. The percentage was very high in 1979--over 90%--but when the final 1986 figures are in, the percentage for that year should be about 20%. FDA (FR 44:(171) 51233-51242, 1979) has estimated that about 20% of all dietary lead is from canned foods and that about two-thirds of this is from lead soldering; therefore, about 14% of all dietary lead originates from lead seams. Recent data provided to FDA by the National Food Processors Association (NFPA) (1986) indicate about a 77% reduction in lead from canned food during the period 1980-1985. This table does not include imported canned foods; we have no data for this contribution to lead in food.

TABLE IX-5. PERCENTAGE OF LEAD-SOLDERED CANS IN ALL U.S. MANUFACTURED FOOD CANS FROM 1979-1985[a]

Year	Total Food Cans (M)	Lead-soldered Cans (M)	Percent of Total
1979	30,543	27,576	90.29
1980	28,432	24,405	85.84
1981	27,638	20,516	74.23
1982	27,544	17,412	63.21
1983	26,942	13,891	51.56
1984	28,121	11,683	41.55
1985	27,767	8,769	31.58
1986[b]	6,517	1,807	27.72

[a] Source: Can Manufacturers Institute data to U.S. FDA., M = Millions.

[b] First quarter, 1986.

FDA activities, to a large extent, consist of establishing voluntary cooperation from domestic food manufacturers and processors, and much of the data are provided by the industry. Undoubtedly, lead in food due to leaching from leaded sources has been significantly reduced. But FDA does not monitor the lead content of imported canned foods, and these imports may have captured significant shares of the domestic market for some food items.

Some changes in steps causing the lead contribution from the food processing industry were not taken until after 1981/1982. In the period

1980-1985, lead in canned food was reduced 77% (NFPA, 1986) and lead in infant foods was reduced considerably (Jellinek, 1982). Recent data from FDA update the age-dependent reduction found in data from the Total Diet Study between 1982-1984 and 1984-1986. Table IX-6 gives total diet lead changes with the percentage decline for some age-sex categories.

TABLE IX-6. AGE- AND SEX-DEPENDENT DIET LEAD INTAKES (μg/kg/day) IN THE UNITED STATES AT TWO TIME PERIODS[a,b]

Age-Sex[c] (Body Weight)	1982-1984 (μg/kg/day)	1984-1986 (μg/kg/day)	Change (%) (μg/kg/day)
6-11 Mo. (9 Kg)	1.70	1.11	-0.59 (35)
2 yr. (13 Kg)	1.60	1.00	-0.60 (37)
14-16 F (54 Kg)	0.48	0.30	-0.18 (33)
14-16 M (60 Kg)	0.63	0.38	-0.25 (40)
25-30 F (60 Kg)	0.43	0.27	-0.16 (39)
25-30 M (76 Kg)	0.48	0.29	-0.19 (40)
60-65 F (64 Kg)	0.42	0.25	-0.17 (40)
60-65 M (76 Kg)	0.44	0.26	-0.18 (42)

[a] Source: FDA Division of Toxicology, Communication of Internal Tabulations to ATSDR, April 23, 1987; based on Total Diet Study results.

[b] Revised Total Diet Study points, 8 collections.

[c] Last six age-sex entries are in years.

The data for the ongoing Total Diet Study are based on samples that are very small in relation to the enormous quantities of food units produced and consumed in the United States and probably do not account adequately for variation by region and multiplicity of processors. The types of food items selected for testing also may not reflect the variations in food selection and consumption patterns among various segments of the U.S. population. The level of lead in food may, consequently, be smaller or greater than indicated.

2. Primary Prevention Exposure Using Combined Environmental and Biological Measures

Biological factors can suppress lead uptake into the body or enhance its excretion. When these factors are nutrients that have well-established interactive relationships with lead uptake and toxicity, such nutrients can be used to reduce internal or *in vivo* exposure. Such factors, when employed in a prophylactic, communitywide way, can also be viewed as an example of primary prevention. When these factors are exploited on an ad hoc basis in children or families where lead poisoning has occurred, their use becomes more a secondary prevention measure. Chapter III discusses metabolic interactions of lead.

As discussed by U.S. EPA (1986a) and Mahaffey et al. (1986), a number of nutritional factors suppress lead absorption and toxicity in test animal and human populations. However, only a few, particularly iron and calcium, can realistically be considered for preventive community medicine for high-risk populations.

Results of numerous studies have shown that both calcium status and iron status in young children are inversely related to the lead absorption level--that is, as calcium or iron levels decrease, lead levels rise. Most of these studies are discussed in U.S. EPA (1986a). A more recent analysis of the NHANES II survey data showed a significant negative correlation between calcium status and Pb-B levels in a group of children under 11 years of age (Mahaffey et al., 1986). As Mahaffey (1982) has indicated, improving the nutritional status of children with high risk of exposure/toxicity greatly increases the effectiveness of environmental lead abatement. But nutritional supplements only shift the lead level required for toxicity rather than eliminating lead uptake and its effects entirely.

Other antagonizing nutrients may not be particularly useful or advisable in this connection. Levels of phosphorus in most diets seem high enough to suggest intake is at adequate levels in poorer children, which is borne out by the Mahaffey et al. (1986) examination of the NHANES II data for children. Vitamin D enhances lead uptake in the gut, but its intake is essential to health and cannot be reduced. As noted in comments on active versus passive measures in the Introduction, nutrition monitoring and maintenance are probably best done in relation to the lead antagonizing nutrients in a program of overall nutritional care, that is, the Women, Infants, and Children (WIC) nutrition program. The level of funding and other support for such programs determines their potential in reducing net lead exposure. We can, in fact, reverse the issue and say that increased nutritional impairment for those at high risk for lead poisoning will enhance exposure and toxicity risk in that population.

B. SECONDARY PREVENTION MEASURES FOR LEAD EXPOSURE

This section assesses environmental, environmental/biological, and extra-environmental measures.

1. Environmental Lead Control

This discussion addresses screening programs and other aspects of early intervention in exposure and toxicity, and environmental hazard identification and hazard abatement.

a. Screening Programs and Case Finding

The 1971 Lead-Based Paint Poisoning Prevention Act, as noted by Farfel (1985), did not specifically dictate health-based (secondary prevention) versus hazard-abatement (primary prevention) steps to be taken to ameliorate lead poisoning in U.S. children. While Title II of the Act authorized grant appropriations to the responsible agency to remove leaded paint on a tract basis in high-risk neighborhoods, funding for this purpose was not actually provided. The Department of Health, Education, and Welfare emphasized intervention--including medical management if necessary--for documented toxicity.

The various screening programs, their history, and their quantitative aspects, were discussed in Chapter V; the focus here is on their role as secondary prevention instruments. While the screening programs were administered by the U.S. CDC (until FY 1982 when CDC control ended) about 4 million children were screened nationwide, and about 250,000 children were registered as having met toxicity risk criteria. The screening program surveyed about 30% of the high-risk children. The detection rates for positive toxicity are considerably below those found by NHANES II, for reasons noted in Chapter V. Case finding and cluster testing, followed by targeted screening, also produce much higher positive response rates (Farfel, 1985).

Screening and early detection of exposure and toxicity undoubtedly have reduced the rates of severe lead poisoning. However, chronic exposure and lower grade toxicity appear more resistant to such secondary prevention approaches. The persistence of these problems is predictable, given the levels and types of unabated exposure remaining in the United States.

In 1981, Federal resources for screening were put under the program of the Maternal and Child Health Block Grants to States. Although the States' use of Federal funds for lead screening programs was estimated by one source to have been reduced initially by 25% (Farfel, 1985), a precise figure cannot be readily given since allocations of the block grant funds for particular projects are determined by the States according to their priorities, and data are not systematically collected on these State funding allocation decisions.

The evidence of the national impact of this initial reduction in Federal resources appears to be mixed. While it appears that the total number of screening program units in the nation has decreased from 60 to between 40 and 45 (Chapter V), there is also evidence in some States and localities that the number of children currently being screened has increased since 1981 (CDC, 1982; Public Health Foundation, 1986). However, a study of data from Newark, NJ for a nine-year period prior to implementation of the block grants showed that the rate of high-lead exposures in asymptomatic children increased about fourfold after funding for lead screening and public education programs was reduced (Schneider and Lavenhar, 1986). Based upon this report, it is likely that those areas that choose to decrease the efficiency of their lead screening services can expect to experience increases in the number of children with lead poisoning.

A key point in the Schneider and Lavenhar (1986) report and additional information given below is that screening programs, especially those supported at a level that allows blanket screening, are particularly cost-effective. This is demonstrated by comparing data on the costs of treating children who are poisoned because early lower levels of lead intoxication were not detected by screening with the costs of community screening programs. According to O'Hara (1982), the cost of repeat admissions to Baltimore hospitals for 19 lead-poisoned children in 1979 was $141,750, or at least $300,000 in 1986 dollars. In summary statistics submitted to ATSDR for the 1985-1986 program year, St. Louis listed budgetary support of $303,453 from the city and $100,000 from the State of Missouri. During that funding year, all agencies in the St. Louis program tested 12,308 children, of whom 1,356 or 11.02% tested positive for lead exposure using CDC classifications. Of these positives, 849 tested as Class II, 445 as Class III, and 62 as Class IV, the most severe level of toxicity. The new CDC guidelines of 1985 were implemented midway through the 1985 screening year (July 1, 1985), so these figures represent a low boundary for the number of positives.

In St. Louis, the entire screening program cost $403,453 and identified 1,356 cases of toxicity; that is just under $300 per poisoned child. In Baltimore, the multiple hospital admissions required for only some of the poisoned children cost about $16,000 per child in 1986 dollars. This does not account for additional essential costs for adequate management of severe toxic cases. These additional huge costs stem from medical follow-up care and treatment, remedial education, etc. The monetized costs of the sequelae in significant toxicity cases are spelled out in U.S. EPA (1985 and 1986b). The effectiveness of screening children for lead poisoning is well demonstrated in terms of deferred or averted medical interventions, and in most settings, is quite cost-effective.

In March 1987, the Committee on Environmental Hazards, American Academy of Pediatrics, issued its "Statement on Childhood Lead Poisoning." It includes this statement:

> "...to achieve early detection of lead poisoning, the Academy recommends that all children in the United States at risk of exposure to lead be screened for lead absorption at approximately 12 months of age.... Furthermore, the Academy recommends follow-up...testing of children judged to be at high risk of lead absorption."

These guidelines from America's pediatric medicine community probably cannot be effectively implemented or coordinated with the current levels or existing type of program support at local, State, and Federal levels.

b. Environmental Hazard Identification and Abatement for Severe Poisoning Cases

When cases of toxicity were found, mass screening programs for lead poisoning routinely made efforts to find the sources. A careful examination of the information on reducing lead exposure by completely or partially removing leaded paint clearly shows that, at best, the effect is debatable. At worst, the approach may not work. In a prospective study, Chisolm et al. (1985) observed that when children return to "lead abated" structures, their Pb-B levels invariably return to unacceptable levels. This is not a case of endogenous Pb-B increase from the release of bone lead, because children heavily exposed before treatment will respond better when placed in lead paint-free housing.

Ample information has accumulated to show that leaded paint removal is hazardous to the workers doing the removal and that lead from the paint continues to be hazardous to the occupants because residual material has been moved to other areas that children contact. A major difficulty is the relative mobility of powdering lead paint, which enters cracks and crevices settles on contact surfaces, and readily sticks to children's hands. As Charney et al. (1983) noted, response to the dust problem may well be as effective as removing the paint film.

The problem of continued exposure risk, even during or after leaded paint abatement, can be illustrated in the recent study by Rey-Alvarez and Menke-Hargrove (1987). Rey-Alvarez and Menke-Hargrove examined a total of 13 lead-poisoned children whose exposure had been exacerbated in varying ways when leaded paint was being or had been removed. In the case of a child remaining in a unit where lead paint was being removed, the child's Pb-B abruptly increased from an average of about 45 µg/dl to 130 µg/dl at the end of the paint removal period. In 12 other children, Pb-B levels that were already elevated increased to higher levels after lead abatement of the unit's interior surfaces. The erythrocyte protoporphyrin levels also greatly increased. Farfel and Chisolm (1987) also document that traditional paint removal increases household dust and child Pb-B levels. The Rey-Alvarez and Menke-Hargrove (1987) and Farfel and Chisolm (1987) data augment the experiences of other investigators and make it clear that lead exposure during and after paint lead removal can be only marginally lowered and may actually increase.

Chisolm (1986) has drawn attention to the need for some fresh approaches to the problem of removing lead from occupied housing. One potentially promising technique is a "wet" method for removing leaded paint from surfaces, which eliminates the creation of lead dust, but it requires extensive field testing. The key to removal is retention and control over the material being removed. Extensive work in this area is required to identify safe and effective methods.

Finally, a hidden assumption underlies the efforts to remove leaded paint from the homes of children found to have lead poisoning: residential stability--that the child will remain in the cleaned-up home. In reality, there is high residential mobility among poor, inner-city residents. The long-term effectiveness of unsystematic "spot" abatement is questionable, perhaps even for the individual children for whom the effort has been made.

2. Environmental/Biological Prevention Measures

This approach is analogous to that described for primary prevention strategies that combine nutrition and environmental control. As a secondary prevention measure, however, nutritional optimization might be more debatable than when it is used on a community level with children not already showing signs of lead toxicity. A secondary nutritional approach would also require that the affected family take a more active role, and this raises the issue of compliance, funds for adequate diets, etc.

3. Extra-Environmental Prevention Measures

These measures are legal sanctions to force the removal of lead from documented poisoning sites. Legal sanctions are some of the tools available for addressing demonstrated and significant health risks. Can one effectively use a legal framework to expedite the rapid and safe removal of lead hazards from children's daily environment? Conversely, can we conclude that a real handicap for such action is the absence of supporting legal tools?

Finding answers to these questions in the available information is not easy, but it is useful to examine a screening program with a legal component and assess its contribution to overall abatement. In its summary of screening activities submitted to ATSDR, the City of St. Louis summarized its dealings with landlords and others who own housing or public-use facilities where lead poisoning had been found. A summary of 1985 court activity stemming from lead hazards, including the licensing of day care centers and similar institutions, indicated a case load of 1,086, with 387 of the cases carried over from 1984. From this cumulative docket, 154 defendants were fined $2,447, an average of $16. Minor fines appeared to be the only measure at the city's disposal, because the 1984 count was virtually identical to 1985's and the average fine for 1984 was the same as for 1985. We cannot say whether minor fines as legal sanctions influenced the city's lead toxicity rate as identified from screening. In the most recent data, this rate was 11%--a rate that has remained about the same since 1978. This case study does suggest, however, that the persisting high lead toxicity rate has not resulted in more effective legal measures.

X. A REVIEW OF ENVIRONMENTAL RELEASES OF LEAD AS EVALUATED UNDER SUPERFUND

Section 118(f)(2) of the Superfund Amendments and Reauthorization Act (SARA) of 1986 requires this report to "score and evaluate specific sites at which children are known to be exposed to environmental sources of lead due to releases, utilizing the Hazard Ranking System of the National Priorities List." EPA has carried out this requirement in two ways: (1) by identifying proposed and final sites on the National Priorities List (NPL) that have been numerically scored under the Hazard Ranking System (HRS) and at which lead has been released into ground or surface waters or air, highlighting those sites where children are known to have been exposed to lead; and (2) by gathering data at an urban area in Boston where children are known to be exposed to lead in soil, and scoring one residence as a site under the HRS.

The HRS was designed to respond to section 105(a)(8)(A) of the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA). This section requires that the National Contingency Plan (NCP) include "criteria for determining priorities among releases or threatened releases throughout the United States for the purpose of taking remedial action,...." Section 105(a)(8)(B) requires that the criteria be used to prepare a list of national priorities for sites with known or threatened releases of toxic substances throughout the United States. This use of the HRS to form the NPL is a means of directing EPA response resources to those facilities believed to present the greatest magnitude of potential harm to human health and the environment.

The HRS is a means of comparing one site against others based on the estimated relative threat to human health and the environment. Relative threat is determined by assessing the likelihood of release or migration of waste contaminants from a facility, along with the consequences of such a release, such as effects on people. Migration of contaminants from a site occur through air, surface water, and groundwater. Consequences of such a release are determined by the toxicity and other characteristics of the wastes and whether the release has affected or will affect people.

The HRS is not an assessment of the risks found at a facility. Such an assessment occurs only after a great deal of additional data have been gathered and is used to help determine the type and degree of cleanup necessary to reduce the risk to human health to an acceptable level.

A. NPL SITES--PROPOSED AND FINAL

To be listed on the NPL, a site must score at least 28.5 out of a possible score of 100 under the HRS in effect September 30, 1987. (EPA is now revising the HRS; see discussion later in this chapter.) Of the 957 proposed and final NPL sites as of September 30, 1987, 307 have lead as an identified contaminant, and 174 have an observed release of lead to air, to surface water, or to groundwater. An observed release is documented by monitoring data showing such a release from the site. The sites with only an identified contaminant had no data showing release of the contaminant from the site. All proposed and final NPL sites with an observed release of lead are listed (with their HRS scores) in Appendix F.

Exposure of Children to Lead at NPL Sites

EPA reviewed site files to obtain data regarding the exposure of children at each NPL site with an observed release of lead. In only a few cases was the Agency able to document exposure of children to lead from the site, since no records are kept that separate children from the general population exposed to releases from a site. In some cases, however, studies had been conducted around sites that showed that children were exposed to lead from the sites. These are documented below.

The Interstate Lead Company, an NPL site in Leeds, AL (HRS score 42.86), is a battery recycling and secondary lead smelting operation. Results of a March 1984 study of lead contamination conducted by the Jefferson County Department of Health and Bureau of Communicable Diseases, showed that children under 10 years of age living less than one-half mile from the lead plant had higher blood lead levels than children the same age living farther from the plant. Blood lead levels of all children ranged from 6 to 29 μg/dl.

The East Helena, MT, site (HRS score 61.65) is a primary lead and zinc smelter where 8.4 square miles of land have been contaminated, with lead in soil measuring more than 1,000 ppm. In a random sample of 90 children living near the smelter, blood lead levels of 6 to 25 μg/dl were detected. In 1975 and 1978, cattle reportedly died from lead poisoning.

At the NL Industries/Taracorp Lead Smelter, an NPL site in Illinois (HRS score 38.11), the Illinois EPA measured high lead levels in the soil of residential areas near the smelter. Two soil samples exceeded 5,000 ppm lead. The Illinois EPA recommended that small children living nearby be restricted from playing in the dirt, from eating outside, and from placing dirt or dirty objects in their mouths.

The Sharon Steel Smelter, in Utah, has been proposed for the NPL (HRS score 73.49). It is an inactive smelter with 10 million tons of tailings piled on the site. People have taken some of the waste from these piles to use in sandboxes and gardens. Analyses by the State of Utah indicate elevated levels of lead and other heavy metals in edible portions of food grown on soil to which the waste from this site has been added.

At the Harbor Island Battery Recycling site in Washington State (HRS score 34.60), elevated levels of lead have been reported in workers and their children.

At the Brown's Battery Recycling site in Pennsylvania (HRS score 37.34), the Pennsylvania Department of Health measured elevated blood lead levels in four children. One child received treatment to reduce his body burden of lead. Soil from three residences adjacent to the primary disposal area had lead levels ranging from 1,120 to 84,200 ppm.

At the Bunker Hill Mining and Metallurgical site (HRS score 54.76), a lead smelter in Idaho, the Idaho Department of Health and Welfare found an epidemic proportion of children (98%) living within 2 miles of the smelter who had blood lead levels exceeding 40 μg/dl.

The Lackawanna Refuse site in Pennsylvania (HRS score 36.57) is a former strip mining site. It is near a residential area of 9,500 people, and local children use the site as a recreational area. EPA and the Pennsylvania Department of Environmental Resources found concentrations of 12,000 ppm lead in the waste contained in the thousands of drums found at the site.

As can be seen from the descriptions of the foregoing cases, the sites on the National Priority List with observed releases of lead consist of sites where manufacturing, processing, or disposal of lead has taken place. This can be seen graphically in Table X-1, relating the twelve most common activities at NPL sites with observed lead releases from waste. Actual disposal of waste accounts for the largest proportion by number of sites, with manufacturing, ore processing, and battery recycling at the bottom of the list.

B. URBAN AREA SITE

EPA has carried out a preliminary assessment and site investigation of a Boston area to prepare an HRS package for scoring the site. The site consists of a rectangular area encompassing approximately 5 square miles where children are believed to be exposed to lead from both interior and exterior paint and from elevated lead concentrations in the soil surrounding the houses. This site was chosen because it contains areas that have been designated by the City of Boston as Emergency Lead Poisoning Areas (ELPAs). An ELPA is an area of one or more city blocks where a higher than average number of children were found to have elevated blood lead levels.

The areas consist mostly of triple-story houses of frame construction. Most have been converted to six apartments, causing a high population density in the area. Because the houses are separated by only a few feet, lack of sunlight inhibits the growth of grass or a suitable cover for the soil, and the lead is available to children playing in the dirt in these areas.

Data were gathered for two housing units within the area specifically to be evaluated under the HRS. Scoring was done for the unit expected to score highest under the HRS. This unit has greatly elevated soil lead levels both in front and behind the house, but no evidence of peeling paint. The lead concentrations in the front of the house near the street exceed the concentrations in the back of the house, indicating that a portion of the lead may have resulted from auto emissions.

The data for this area have been processed through the Hazard Ranking System as if it were a hazardous waste disposal site to be evaluated for the NPL. The data were collected by Region I personnel and scored prior to transmission to headquarters. This original scoring package passed through the quality assurance and quality control process without revisions, and the assigned score of 3.56 was affirmed. The minimum HRS score needed for listing on the NPL is 28.5.

TABLE X-1. **MOST COMMON ACTIVITIES ASSOCIATED WITH LEAD WASTE AT NPL SITES WITH LEAD RELEASE**[a]

Activity of NPL Site	With Observed Lead Release		All NPL Sites		
	Rank	Number of Sites	Rank	Number of Sites	Percentage With Lead Release
Landfill, commercial/ industrial	1	75	2	349	21
Surface impoundments	2	66	1	350	19
Containers/drums	3	49	3	261	19
Landfill, municipal	4	35	4	158	22
Waste piles	5	27	10	89	30
Spill	6	21	6	139	15
Other manufacturing/ industrial	7	18	5	142	13
Chemical process/ manufacturing	8	18	7	104	17
Battery recycling	9	15	24	17	88
Tank, above ground	10	14	9	94	15
Leaking containers	11	10	8	95	11
Ore processing, refining, smelting	12	9	19	29	31

[a] The release of lead is not necessarily attributed to the specific activity indicated. Sites often have more than one activity and EPA reporting requirements do not identify the activity to which the release of a specific substance is attributable. These activities are present at 162 of the 167 sites with an observed release of lead.

HRS Scoring at the Boston Urban Site

Three pathways in which a contaminant can migrate from a site (the principal criteria for placing a site on the NPL) were evaluated. These include the groundwater pathway in which a contaminant leaches through the soil and into the groundwater, the surface water pathway in which a contaminant is washed into a stream or pond or other surface water, and the air pathway in which a contaminant is volatilized, or otherwise migrates from the site into the air. Each pathway is scored by one of two methods, an observed release, where quantitative evidence exists to show that a contaminant has migrated from the site, or in cases where no quantitative data exist to document an observed release, the potential for release is scored.

Groundwater Pathway. The concern for contaminant migration to groundwater is a concern for drinking water, specifically the health concern for the exposed population drinking water from a contaminated aquifer. In this case, no drinking water aquifer is near the site, and therefore no chance that lead could migrate from the site and contaminate drinking water. In any case, lead tends not to migrate through soil, but rather tends to remain in the topmost centimeter. Since no observed release exists, the potential for lead migration to groundwater must be scored.

Scoring a potential release consists of two parts: route characteristics and containment. If all sections of these two parts are assigned maximum scores, the potential contamination score could equal the score for an observed release. In most instances, the potential for release is assigned a lower score than an actual release.

Route characteristics consist of four physical characteristics of the site and the waste that would indicate a potential for contaminant migration to groundwater. These are: depth to the aquifer of concern, net precipitation, permeability of the unsaturated zone (impediment to migration), and physical state of the waste. Well logs for the Boston study area indicate that depth to water is usually less than 20 feet, resulting in a score of 6, the highest score for that category. The net annual precipitation (rainfall minus evaporation) for the area is 22 inches, resulting in the highest score of 3. The soil in the area is glacial deposit, which is moderately permeable, allowing a score of 2 (of a possible 3). The physical state of the waste is particulate form, but in a powder or fine dust, allowing a score of 2 (of a possible 3). The total score for route characteristics for groundwater contamination is 13 (of a possible 15).

Containment refers to any means (such as grass or ground cover in this case) that would inhibit the migration of the contaminant from the site. Since lead remains in the topmost centimeter of soil, and at the two houses selected for ranking no ground cover was observed, the maximum score of 3 was assigned, meaning no containment was observed.

The score for potential lead migration to groundwater received a score of 39, 6 points below the maximum score of 45 for an observed release. The HRS next evaluates the consequences of such a release in terms of toxicity, persistence and amount of the waste, and the possibility that people might use a potentially contaminated aquifer for drinking water.

A waste characteristics score of 19 -- out of a possible 26 -- was obtained for the site since lead's score of toxicity is set at 18, and the quantity present was scored as 1. The quantity of lead deposited at the site is unknown, but in any case would be a small amount compared with, e.g., waste deposit quantities at dumpsites.

In scoring the possibility that people might use a potentially contaminated well or aquifer for drinking water, the HRS scores the use of the nearest aquifer of concern within a 3-mile radius of the site, the distance to the nearest well that draws water from the aquifer of concern, and the number of people drinking water from wells within a 3-mile radius of the site.

In this case, there is no drinking water well within a 3-mile radius of either site, but there are industrial facilities with wells for industrial purposes within the 3-mile radius. For this reason, a value of 1 (rather than 0) has been assigned. However, no value other than 0 could be assigned as distance to aquifer of concern, and number of people drinking water from wells within a 3-mile radius. This category score is 3 (of a possible 49).

Surface Water Pathway. Scoring for migration of contaminants from a site via the surface water pathway closely follows that of the groundwater pathway. Where no observed release can be documented, as here, the potential release is scored using route characteristics and containment.

Route characteristics for surface water migration include facility slope and intervening terrain to the nearest downhill surface water, rainfall, the distance to the nearest surface water, and the physical state of the waste. In this case, the only surface water near either site is a pond on a golf course, approximately 1,500 feet from one site. Although the slope was less than 1%, a value of 1 was assigned for that factor because the area is highly urbanized, and there are probable paths for surface water flow, such as through storm sewers. The 1-year, 24-hour highest rainfall for the area is approximately 2.5 inches, resulting in a score of 2. The distance to the pond is approximately 1,500 feet, also resulting in a score of 2. The physical state of the waste, namely particulate, is fairly easily transported by rainfall to surface water; therefore the score of 2 was assigned. The route characteristics score is 9 (of a possible 15), which multiplied by the containment score of 3 (of a possible 3) equals 27 (of a possible 45). Waste characteristics would be the same as those scored for groundwater migration, so the score of 19 is the same as that for the groundwater pathway.

The category for potential impact on people or the environment consists of three factors: the use of the water, distance to a sensitive environment, and population served by drinking water intakes downstream from the site. The use of the pond, since it is in a city park, is considered to be recreational, and therefore a value of 2 was assigned. There are no sensitive environments or drinking water intakes near the site; therefore a score of 0 was assigned for each of these factors. The total category score is 4.78.

Air Pathway. The air route for migration of a contaminant from a site can only be scored for an observed release. To document an observed release, it must be shown that lead is migrating by air from the site or has done so in the past. To document an observed release, alternative sources for the lead must be screened out, which means that background levels must be determined so that the lead migrating from the site can be attributed to the site (e.g., lead-based paint). Lead is ubiquitous to urban areas, being released from automobile emissions and from various industries. Most of the lead released settles on soil or pavement. Separating lead attributable to house paint from lead attributable to other sources requires very sophisticated sampling and analytical techniques.

If the lead in soil were shown to be attributable to leaded paint, the lead must also be shown to have migrated through the air. Thus, other migration routes such as physical transfer by the daily activities of residents (e.g., transfer of house dust to soil or removal of paint chips) or transfer by rainfall must be ruled out. Finally, an observed release must be monitored at the breathing zone and show levels significantly above background levels. The

EPA considered methods to obtain such data, but would have had to mount a research project without any assurance of obtaining usable data. The Agency decided to treat this site like any other site to be scored under the HRS, thereby foregoing the research monitoring project. The site score for the air pathway was 0.

Site Migration Scoring. The Site Migration Score is the principal basis for proposing that a site be included on the NPL. It is a composite of the migration scores calculated for each of the three migration pathways. It may be defined as the square root of the sum of the squares of each pathway score divided by 1.73. Sites with a migration score of 28.5 or higher qualify for inclusion on the NPL. The highest Site Migration Score for the two actual Boston urban lead sites was 3.56.

Additional Modes of Potential Harm. Two additional modes of potential harm to people or the environment from a site are also scored under the HRS, but are not used for the purpose of placing a site on the NPL. These modes are direct contact with contaminants from a site and threat of fire or explosion from a site. These scores may be used for purposes of removal actions or for enforcement actions against a responsible party. A site need not be listed on the NPL for EPA to take either removal or enforcement action to protect people or the environment.

Direct Contact Mode. A site may be scored on the basis of an observed incident or on the potential for an incident, using factors of accessibility and containment. In the case of the Boston urban sites, the score was based on an observed incident, namely one documented child with an elevated blood lead level. The score is 45. The waste toxicity was scored at the highest value, 15, for lead. Exposed population within a 1-mile radius is nearly 12,000 people according to U.S. Census data, and this resulted in a score of 4. The distance to a critical habitat is considerably more than one mile, resulting in a score of 0. For the direct contact mode, this site would score 50, a level at which the Agency would reevaluate the site for potential removal action.

Fire and Explosion Mode. The fire and explosion mode is evaluated when either a state or local fire marshal has certified that the site presents a significant fire or explosion threat to the public or to sensitive environments, or a fire and explosion threat has been demonstrated by observation at the site. Such a threat could result in consideration for a removal action at a site. Since neither of these events is relevant to the threat from this urban site, no evaluation of the fire and explosion mode was done.

C. REVISION OF THE HAZARD RANKING SYSTEM

The Superfund Amendments and Reauthorization Act specifically directed EPA to modify the HRS so that "to the maximum degree feasible, it accurately assesses the relative degree of risk to human health and the environment posed by sites." EPA was specifically directed to assess human health risks associated with actual and potential surface water contamination, specifically considering recreational use of the water, and the migration of a contaminant to downstream sources of drinking water; damages from an actual or a threatened release to natural resources that may affect the human food chain; actual or potential ambient air contamination; and those wastes described in Section 3001 of the Resource Conservation and Recovery Act (e.g., flyash, bottom ash, slag waste, and flue gas emission control waste). EPA was also directed to give high priority to facilities where a release has resulted in closure of drinking water wells or has contaminated a principal drinking water supply.

In an Advance Notice of Intent to Revise the HRS, published April 9, 1987, EPA noted that the direct contact mode has been one of the most significant factors in selecting a remedy when cleaning up hazardous waste sites where direct contact with the site and the contaminants were factors. The Agency intends to include a direct contact factor in its revised HRS, for purposes of placing sites on the NPL, and solicited comments on appropriate methods to do so.

EPA is addressing the concerns listed in SARA in its update of the Hazard Ranking System. The choice of data and of models to evaluate the data for each pathway has been reviewed by various offices within the Agency, and by the Agency's Science Advisory Board. An updated version of the HRS is expected to be proposed for comment in the summer of 1988. Comments will be considered and a final update of the HRS will be published.

D. SUMMARY

As pointed out in this chapter, there is no question that children are being exposed to lead at sites that are already on the National Priority List for Superfund remedial action. These sites were placed on the NPL according to their HRS score, which showed migration of lead and other contaminants from the sites, and in some cases actual exposure of children to the lead and other contaminants from the site.

The scoring of the Boston urban lead site under the HRS pointed out the differences of this area from the usual sites scored under the HRS. The Boston site was heavily residential, rather than an abandoned dumping area. It had only one known contaminant rather than an unknown mixture, so that it needed no complex assessment or site inspection. It had no hazard from migration of the contaminant, but rather from the nonmigration of the lead, which made it available to children.

The only mode by which this site could have scored high enough to be placed on the NPL was by the direct contact route. The air route could have contributed to the score if an experimental monitoring project had been conducted and if all conditions had been ideal. This includes enough wind so that the soil containing lead had been blown off site, into the breathing zone of children, and recorded by the monitor.

The revised HRS to be proposed in 1988 is to contain a revised direct contact mode for use in listing NPL sites. Under the new HRS, it is possible that an urban site having leaded paint contamination would score above the HRS cutoff score based on direct contact alone. The proposed HRS will also include a potential air pathway, which, depending on its characteristics, may be more favorable to this sort of site than is the present observed air pathway. This factor may also increase the HRS score of an urban lead-contaminated site.

When the revised HRS is finally available, EPA will be able to determine how such urban lead-contaminated sites will score relative to other sites scored under the system. During the interim, EPA will be collecting data through the pilot projects under Section 111 of SARA to determine the impact of lead-contaminated soil removal on children's blood lead levels.

XI. LEAD EXPOSURE AND TOXICITY IN CHILDREN AND OTHER RELATED GROUPS IN THE UNITED STATES: INFORMATION GAPS, RESEARCH NEEDS, AND REPORT RECOMMENDATIONS

In the preparation of this report to Congress, exposure and health risk problems were identified that require further analysis because of significant gaps in information. These gaps are identified as are research needs that must be addressed in order to fill the gaps. The current status of the lead problem and the underlying data base prompted the recommendations presented.

A. INFORMATION GAPS

As noted early in the report, comprehensive, current, and accurate data on the numbers of children and other groups exposed to lead at some level of concern, by location or source, were not readily available. Since these data did not always exist in the exact form required, we often estimated the numbers, using the statistical techniques that seemed most precise, to be reasonably responsive to the letter and spirit of Section 118(f) of SARA.

Part of this gap in information is being addressed. The National Center for Health Statistics is preparing to carry out the third National Health and Nutrition Examination Survey (NHANES III); information of the type presented in the second survey, with respect to Pb-B and EP levels, will be updated and future Pb-B levels will be collected in ways more useful for various public health purposes. This survey will be executed and analyzed in the period 1988-1994. The 1990 Census will also provide enumerations of children for the different categories for the year 1990. Such data will be more current for such factors as the effects of economic dislocations occurring that are structural rather than cyclical in nature, e.g., shifts from well-paid industrial jobs to services jobs for certain segments of the population. Those data collections will occur well into the future, however, and the estimates in this report will have to suffice as reasonable interim assessments.

We tried several approaches to identify and rank children with elevated Pb-B levels by regional or other division of geographic area, but could not do so in ways that assured a satisfactory level of certainty for the numbers. We therefore provided national-level estimates.

There is surprisingly little information on the actual numbers of young children with a record of being exposed to a source of lead sufficient to raise the Pb-B levels to either some measured amount or to some toxicity level. Information that is available is diffuse in terms of the levels of exposure, the number of years in the childhood age span, and other related measures.

In some cases, exposure is defined according to the number of children potentially at increased risk because of their proximity to lead-emitting sources. In other cases, exposure is estimated on the basis of elevated Pb-B levels. Pb-B levels as risk thresholds are defined differently among studies. However, even potential exposure of children due to contact with a lead source presents a higher risk situation than does the absence of such sources.

Better enumeration and screening for Pb-B levels are required in communities contiguous to stationary lead operations, e.g., primary and secondary smelters. A major problem with estimating the effects of lead in dust and soil as a source for childhood exposure is that there is simultaneous direct exposure to lead from paint and air sources, the primary contributors to this pathway. Better data are needed on the relationships between airborne lead and lead from dust or soil, especially on changing airborne levels. For example, we could not distinguish between children exposed to the two media and encountered overestimating and multimedia-uptake problems. On the other hand, some sizable fraction of these children have other source exposures.

More specific information is required on the distribution of lead concentrations in the tap water of households with young children. U.S. EPA (1986b) has estimated that 20% of households have drinking water above the proposed standard for lead. Assessing actual lead levels in various tap water sources, beyond the projected water-based Pb-B changes given in Chapter VI, would still be helpful.

Information on dietary lead intakes by infants and toddlers needs to be updated. Information is also needed on the intake distribution among children. The model of food consumption by Beloian described in Chapter IX is relevant to earlier rather than more recent food lead levels. The FDA has provided recent in-house estimations of dietary lead intakes and changes therein. Evaluating the efficacy of remaining further actions and future assessment of this exposure route is needed since we will reach the limits to further food lead controls not too far in the future.

Although much information has been presented on the adverse lead effects in young children and other risk groups, gaps remain in our data base. For example, there are questions as to which biological indicator is appropriate for use at which time frame of child development. It is also important to know which of the early effects of lead have the most useful predictive value for later outcomes and which toxic responses will persist, both into later childhood and in later decades. Some of these questions may be answered through the prospective studies under way in the United States and elsewhere.

The area of lead exposure abatement approaches and strategies is plagued with both qualitative and quantitative unknowns. Information is needed on the full range of lead paint/dust/soil removal options in terms of their technology and costs. In particular, field studies are needed on the relative efficacy of lead removal protocols for large abatement efforts versus small efforts with individual homes or contiguous tracts of housing. As part of recent legislation (SARA, 1986), EPA is now setting up and will be evaluating results from several soil lead abatement demonstration projects in the United States.

Present methods of lead removal from homes and other sites and its disposal are relatively crude. Evidence that the methods appear to endanger abatement workers and occupants alike was presented in the report. Also needed are better methods of preventing dispersal of removed lead from one site to other sites. Chisolm (1986) has suggested wet chemical methods using certain paint surface removal agents, and nonthermal, nondust approaches may be the best ways to proceed, so long as these alternatives are safe and economical.

Knowledge is also lacking about the support approaches for any assault on lead exposure via extra-environmental means. These approaches include maintaining optimal nutrition in risk populations through community-level programs and effective legal infrastructures to enforce compliance with abatement measures and timetables. A good overview also is lacking on how changes in screening program organization have affected the scope and effectiveness of lead screening programs in high-risk areas. Because the societal and monetary cost-effectiveness of screening is documented, it is desirable to know the level of undetected toxicity among screened target populations.

B. RESEARCH NEEDS

A minimum inventory of research needs in areas covered by this report includes:

(1) Continuing and regionally comprehensive studies of quantitative relationships between exposed populations and both their geographic distribution and various lead sources are needed. These include the high-lead sources as well as those provisionally viewed as background or low-level. As part of this effort, data collected by U.S. Census-taking methods should include elements relating to environmental exposure to toxicants.

(2) As a follow-up to (1), further examination is needed of body lead-source lead relationships to include dust/soil versus blood lead and paint lead versus blood lead.

(3) Continued development of quantitative biokinetic/aggregate uptake models is necessary for reliable prediction of body lead burdens which may reduce the need for expensive population surveys.

(4) Both the number and scope of prospective studies of lead exposure and toxicity in U.S. populations should be expanded to build on several ongoing U.S. efforts. Present programs offer valuable public health and scientific information on *in utero* and neonatal lead contact and its consequences.

(5) Cautious examination of the use of improved chelation therapy modalities is required, with emphasis on specificity for lead and minimal side effects.

(6) Further research is needed on the relative strengths and shortcomings of biological indicators of systemic exposure. Of particular importance here are *in vivo* measures of lead accumulation in the mineral tissue of young children as a means of providing a biological record of exposure during periods of maximum vulnerability. Other key areas in lead metabolism deal with uterine lead uptake and deposition versus Pb-B values during pregnancy and such relationships as factors determining whether a fetus sustains toxic injury or not.

(7) In the area of lead exposure abatement, research on several fronts is required:

(a) Field studies on the efficacy of broader lead removal from child environments, e.g., at a tract or neighborhood level or larger. Such efforts would include before-and-after evaluation of Pb-B levels, done with careful statistical and quality control/quality assurance protocols. In response to SARA, EPA is now addressing this problem systematically via three demonstration projects.

(b) Field studies of the type detailed above with designs stratified to permit assessment of how such procedures relate to primary contributors, for example, lead paint versus urban air fallout of lead. Here, also, the EPA demonstration projects will be helpful, if sub-studies are made of these variables.

(c) Further development of field methods is necessary to test lead in various exposure media. Of particular importance are improved _in situ_ methods for lead in painted surfaces.

(d) Assessment of the actual physical removal technology for removal of leaded paint , dust, and the like. A related examination of practical disposal plans is also necessary. As noted earlier, leaded paint consists of an aggregate burden of millions of tons, while other inputs also add up to millions of tons. Therefore, disposal is not inconsequential to the lead abatement problem. Moving the lead may also inadvertently shift exposure to another population. A draft report for a model site in Boston (Appendix E), discusses various scenarios and associated problems.

(e) Further examination of the efficacy of lead screening programs for high-risk populations both for their scope and effectiveness and for the relationship between public financial support of screening and the ability to identify children at risk is needed.

(f) Assessment of the relative costs of effective, if expensive, alternatives to the piecemeal abatement, the piecemeal enforcement, and the piecemeal follow-up for reexposure that appears to be the present status of remedial actions. Is it less expensive, in human and resource terms, to consider such measures as relocation?

(g) Research that explores the feasibility of better biochemical screening measures beyond the use of erythrocyte protoporphyrin (EP) since this measure is not reliable and yields too many false negatives. Failure to detect positive cases makes such research urgent.

C. RECOMMENDATIONS

In view of the multiple sources of lead exposure, an attack on the problem of childhood lead poisoning in the United States must be integrated and coordinated to be effective. In addition, such an attack must incorporate well-defined goals so that its progress can be measured. For example, the lead exposure of children and fetuses must be monitored and assessed systematically if efforts to reduce their exposure are to succeed. A comprehensive attack on the U.S. lead problem should not preclude focused efforts by Federal, state, or local agencies with existing statutory authorities to deal with different facets of the same problem. Indeed, all relevant agencies should continue to respond to this important public health problem, but do so with an awareness of how their separate actions relate to the goals of a comprehensive attack.

The following specific measures are recommended to support the general objective of eliminating childhood lead poisoning.

1. Lead in the Environment of Children

(a) We recommend that efforts be implemented to reduce lead levels in sources that remain major causes of childhood lead toxicity.

(1) Leaded paint continues to cause most of the severe lead poisoning in U.S. children. It has the highest concentration of lead per unit of weight and is the most widespread source, being found in approximately 21 million pre-1940 homes.

(2) Dust and soil lead, derived from flaking, weathering, and chalking paint plus airborne lead fallout over the years, is the second major source of potential childhood lead exposure.

(3) Drinking water lead is of intermediate but highly significant concern as an exposure source for both children and the fetuses of pregnant women. Food lead also contributes to exposure of children and the fetuses.

(4) Lead in drinking water is a controllable exposure source and state and local agencies should be encouraged to enforce strictly the Federal ban on the use of leaded solder and plumbing materials. Stronger efforts should also be made to reduce exposure to lead-based paint and dust/soil lead around homes, schools, and play areas.

(b) We recommend that efforts to reduce lead in the environment be accompanied by scientific assessments of the amounts in each of these sources through strengthening of existing programs that currently attempt such assessment. The largest information gap exists in determining which housing, including public housing, contains leaded paint at hazardous levels. A similar information gap exists for information in distributions of soil/dust lead on a regional or smaller-area basis. Systematic monitoring of lead exposures from food and water is urgently needed. Programs, such as the FDA's Total Diet Study, must be supplemented. The data currently collected with the resources available do not yield sufficient information on the high risk strata of the population to support intervention measures. Water monitoring programs suffer from a paucity of systematically collected data.

(c) Use of precise and sensitive methodologies is essential for environmental monitoring of source specific lead. More sensitive and precise techniques are required for *in situ* field testing of lead in painted surfaces.

(d) Major improvements in the collection, interpretation, and dissemination of environmental lead data on a national basis are required and recommended to assess the extent of remaining lead contamination and to identify trends. Data from screening programs should be compiled nationally and made uniform so that geographic differences in lead toxicity rates can be determined.

(e) The need to examine fully the extent of lead contamination in *all parts* of the child's environment remains. We recommend emphasis on examining the presence of lead in schools, day care centers, nurseries, kindergartens, etc., particularly the lead in paint, in drinking water, and in soil and dust in such facilities.

(1) We recommend that all attempts at source-specific reduction in children's environments be accompanied by assessment of the long-term effectiveness and efficiency of such actions (see Section 2 of Recommendations also).

(2) Lead is both a ubiquitous and persistent pollutant. Planned lead reductions in any environmental compartment must be evaluated in terms of impacts on other compartments so that fruitless shifting of the problem from one source or medium to another is avoided. For example, when leaded paint or soil is removed from a child's environment, ultimate, safe disposal must be considered.

(3) The evidence is strong that *in utero* exposure of the developing fetus occurs at potentially toxic levels in some proportion of pregnant U.S. women. This risk population needs close attention to assess and reduce their most significant lead exposure sources, which should especially include occupational exposures.

(4) Lead pollution is a health problem that involves almost all segments of U.S. society. Extra-environmental or legal measures should be explored to reduce lead levels in the environment by both public and private sectors.

2. Lead in the Bodies of Children

(a) Children are being exposed to and poisoned by lead while environmental lead reduction is under way. Screening programs with sufficient funding to make a real and measurable impact are urgently needed.

(b) There is a need to maintain screening programs extant in some states that currently identify children at risk from lead exposure at or above blood-lead levels of 25 µg/dl. Since current EP tests, used as the initial screen, cannot accurately identify children with blood lead levels below 25 µg/dl, screening tests that will identify children with lower blood-lead levels must be developed.

(c) The 1987 statement of the American Academy of Pediatrics calling for lead screening of all high risk children should be supported by assistance in implementation.

(d) Use of *in vivo* cumulative lead screening methods is recommended as soon as available. A quick, accurate, noninvasive screening test would be better accepted by parents, resulting in many more children being screened.

(e) We recommend that screening be extended to all high-risk pregnant women, with particular emphasis on urban teenaged pregnant women, and that prenatal medical care providers be involved in this effort.

(f) We recommend determination of the prophylactic role of nutrition in ameliorating systemic lead toxicity.

(g) Further use should be made of metabolic models already developed and research to refine them should be done. This will enable their use to predict total body burden contributions from varying environmental sources of known lead levels.

(h) Long-term prospective studies of lead's effects during child growth and development should continue to be supported through appropriate support mechanisms, beginning with the relationship of maternal lead burden to *in utero* toxicity and including children with neurological disabilities and genetic disorders, such as sickle cell anemia.

(i) Nationwide assessments of lead toxicity status in U.S. children on a continuing basis are recommended. Efforts such as the planned NHANES III survey should be supported to maximize the data collected about lead exposure levels. Support as well should be provided for more geographically focused surveys, e.g., on the level of Metropolitan Statistical Areas (MSAs).

REFERENCES

Alexander, F. W.; Delves, H. T. (1981) Blood lead levels during pregnancy. Int. Arch. Occup. Environ. Health 48: 35-39.

Alexander, F. W.; Delves, H. T.; Clayton, B. E. (1973) The uptake and excretion by children of lead and other contaminants. In: Barth, D.; Berlin, A.; Engel, R.; Recht, P.; Smeets, J., eds. Environmental health aspects of lead: proceedings, international symposium; October 1972; Amsterdam, The Netherlands. Luxembourg: Commission of the European Communities; pp. 319-331.

American Academy of Pediatrics. (1987) Statement on childhood lead poisoning. Committee on environmental hazards/committee on accident and poison prevention. Pediatrics. 79: 457-462.

Angle, C. R.; Marcus, A.; Cheng, I.-H.; McIntire, M. S. (1984) Omaha childhood blood lead and environmental lead: a linear total exposure model. Environ. Res. 35: 160-170.

Annest, J. L.; Mahaffey, K. R. (1984) Blood lead levels for persons ages 6 months-74 years, United States, 1976-80. Washington, DC: U.S. Department of Health and Human Services; DHHS publication no. (PHS) 84-1683. (National health and nutrition examination survey series 11, no. 233.)

Annest, J. L.; Pirkle, J. L.; Makric, D.; Neese, J. W.; Bayse, D. D.; Kovac, M. G. (1983) Chronological trend in blood lead levels between 1976 and 1980. N. Engl. J. Med. 308: 1373-1377.

Araki, S.; Ushio, K. (1982) Assessment of the body burden of chelatable lead: a model and its application to lead workers. Br. J. Ind. Med. 39: 157-160.

Arizona Department of Health Services. (1976) Lead-based paint report of findings to the State Legislature, December 20.

Baghurst, P. A.; Robertson, E. F.; McMichael, A. J.; Vimpani, G. V.; Wigg, N. R.; Roberts, R. R. (1987) The Port Pirie cohort study: lead effects on pregnancy outcome and early childhood development. Neurotoxicology 8: 395-402.

Baker, E. L., Jr.; Falland, D. S.; Taylor, T. A.; Frank, M.; Peterson, W.; Lovejoy, G.; Cox, D.; Housworth, J.; Landrigan, P. J. (1977) Lead poisoning in children of lead workers: house contamination with industrial dust. N. Engl. J. Med. 296: 260-261.

Baltimore City Health Department. (1971) Chronology of lead poisoning control: Baltimore, 1931-1971. Baltimore Health News 48: 34-40.

Bander, L. K.; Morgan, K. J.; Zabik, M. E. (1983) Dietary lead intake of preschool children. Am. J. Public Health 73: 789-794.

Barltrop, D. (1969) Transfer of lead to the human foetus. In: Barltrop, D.; Burland, W. L., eds. Mineral metabolism in pediatrics. Philadelphia, PA: Davis Co.; pp. 135-151.

Barltrop, D. (1972) Children and enviromental lead, In: Hepple, P., ed. Lead in the environment: proceedings of a conference. London, United Kingdom: Institute of Petroleum; pp. 52-60.

Barry, P. S. I. (1975) A comparison of concentrations of lead in human tissues. Br. J. Ind. Med. 32: 119-139.

Barry, P. S. I. (1981) Concentrations of lead in the tissues of children. Br. J. Ind. Med. 38: 61-71.

Bayley, N. (1969) Bayley scales of infant development. New York, NY: Psychological Corp.

Bellinger, D. C.; Needleman, H. L.; Leviton, A.; Waternaux, C.; Rabinowitz, M. B.; Nichols, M. L. (1984) Early sensory-motor development and prenatal exposure to lead. Neurobehav. Toxicol. Teratol. 6: 387-402.

Bellinger, D. C.; Leviton, A.; Waternaux, C.; Needleman, H. L.; Rabinowitz, M. B. (1985) A longitudinal study of the developmental toxicity of low-level lead exposure in the prenatal and early postnatal periods. In: J. D. Lekkas, ed. International conference: heavy metals in the environment, v. 1; September; Athens, Greece. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 32-34.

Bellinger, D. C.; Leviton, A.; Needleman, H. L.; Waternaux, C.; Rabinowitz, M. B. (1986) Low-level lead exposure and infant development in the first year. Neurobehav. Toxicol. Teratol. 8: 151-161.

Bellinger, D.; Leviton, A.; Waternaux, C.; Needleman, H.; Rabinowitz, M. (1987a) Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N. Engl. J. Med. 316: 1037-1043.

Bellinger, D.; Sloman, J.; Leviton, A.; Waternaux, C.; Needleman, H.; Rabinowitz, M. (1987b) Low-level lead exposure and child development: assessment at age 5 of a cohort followed from birth. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 49-53.

Beloian, A. (1982) Use of a food consumption model to estimate human contaminant intake. Environ. Monit. Assess. 2: 115-127.

Beloian, A. (1985) Model system for use of dietary survey data to determine lead exposure from food. In: K. R. Mahaffey, Dietary and environmental lead: human health effects. Amsterdam, The Netherlands: Elsevier; pp. 109-155.

Benignus, V. A.; Otto, D. A.; Muller, K. E.; Seiple, K. J. (1981) Effects of age and body burden of lead on CNS function in young children. II. EEG spectra. Electroencephalogr. Clin. Neurophysiol. 52: 240-248.

Billick, I.; Gray, V. (1978) Lead-based paint-poisoning research, review and evaluation, 1971-1977. Washington, DC: U.S. Department of Housing and Urban Development; HUD RP-809.

Blackfan, K. D. (1917) Lead poisoning in children with especial reference to lead as a cause of convulsions. Am. J. Med. Sci. 153: 877-887.

Blake, K. C. H. (1976) Absorption of ^{203}Pb from gastrointestinal tract of man. Environ. Res. 11: 1-4.

Blake, K. C. H. (1980) Radioactive lead studies in the human [dissertation]. Capetown, South Africa: University of Capetown.

Bornschein, R. L.; Rabinowitz, M. B., eds. (1985) The second international conference on prospective studies of lead; April 1984; Cincinnati, OH. Environ. Res. 38(1).

Bornschein, R. L.; Succop, P.; Dietrich, K. N.; Clark, C. S.; Que Hee, S.; Hammond, P. B. (1985) The influence of social and environmental factors on dust lead, hand lead and blood lead levels in young children. Environ. Res. 38: 108-118.

Bornschein, R. L.; Succop, P. A.; Dietrich, K. N.; Krafft, K.; Grote, J.; Mitchell, T.; Berger, O.; Hammond, P. B. (1987a) Pre-natal lead exposure and pregnancy outcomes in the Cincinnati lead study. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 156-158.

Bornschein, R. L.; Succop, P. A.; Krafft, K. M.; Clark, C. S.; Peace, B.; Hammond, P. B. (1987b) Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Hemphill, D. D., ed. Trace substances in environmental health - XX: proceedings of University of Missouri's 20th annual conference; June 1986; Columbia, MO. Columbia, MO: University of Missouri; pp. 322-332.

Bornschein, R. L.; Grote, J.; Mitchell, T.; Succop, P.; Shukla, R. (1987c) Effects of prenatal and postnatal lead exposure on fetal maturation and postnatal growth. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Brunekreef, B. D. (1984) The relationship between air lead and blood lead in children: a critical review. Sci. Total Environ. 38: 79-123.

Brunekreef, B. D.; Noy, D.; Biersteker, K.; Boleij, J. (1983) Blood lead levels of Dutch city children and their relationship to lead in the environment. J. Air Pollut. Control Assoc. 33: 872-876.

Burchfiel, J. L.; Duffy, F. H.; Bartels, P. H.; Needleman, H. L. (1980) The combined discrimination power of quantitative electroencephalography and neuropsychologic measures in evaluating central nervous system effects of lead at low levels. In: H. L. Needleman, ed. Low level lead exposure: the clinical implications of current research. New York, NY: Raven Press; pp. 75-89.

Byers, R. K.; Lord, E. E. (1943) Late effects of lead poisoning on mental development. Am. J. Dis. Child. 66: 471.

Centers for Disease Control. (1978) Preventing lead poisoning in young children. J. Pediatr. (St. Louis) 93: 709-720.

Centers for Disease Control. (1982) Lead poisoning. Morbid. Mortal. Rep. 30: 112-113.

Centers for Disease Control. (1983) Abortion surveillance, 1979-1980. Issued May 1983. (Tables 3, 6, 7, and 8).

Centers for Disease Control. (1985) Preventing lead poisoning in young children: a statement by the Centers for Disease Control, January, 1985. Atlanta, GA: U.S. Department of Health and Human Services; no. 99-2230.

Centers for Disease Control. (1986a) East Helena, Montana, child lead study, summer, 1983: final report. July, 1986.

Centers for Disease Control. (1986b). Final report: Kellogg revisited-1983. Childhood blood lead and environmental status report. Panhandle District Health Department, Idaho Department of Health and Welfare, Center for Environmental Health/Center for Disease Control, Public Health Service, United States Environmental Protection Agency, July, 1986.

Centers for Disease Control. (1986c) Abortion statistics, U.S. 1982-1983 [Prerelease to state health agencies, 1986]. Center for Health Promotion and Education.

Chamberlain, A. C. (1983) Effect of airborne lead on blood lead. Atmos. Environ. 17: 677-692.

Chamberlain, A. C.; Heard, M. J.; Little, P.; Newton, D.; Wells, A. C.; Wiffen, R. D. (1978) Investigations into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority; report no. AERE-R9198.

Charney, E.; Sayre, J.; Coulter, M. (1980) Increased lead absorption in inner city children: where does the lead come from? Pediatrics 65: 226-231.

Charney, E.; Kessler, B.; Farfel, M.; Jackson, D. (1983) Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. N. Engl. J. Med. 309: 1089-1093.

Chisolm, J. J., Jr. (1981) Dose-effect relationship for lead in young children: evidence in children for interactions among lead, zinc and iron. In: Lynam, D. R.; Piantanida, L. G.; Cole, J. F., eds. Environmental lead: proceedings of the second international symposium on environmental lead research; December, 1978; Cincinnati, Ohio. New York, NY: Academic Press; pp. 1-7. (Ecotoxicology and environmental quality series).

Chisolm, J. J., Jr. (1986) Removal of lead paint from old housing: the need for a new approach. Am. J. Public Health 76: 236-237.

Chisolm, J. J., Jr.; Barltrop, D. (1979) Recognition and management of children with increased lead absorption. Arch. Dis. Child. 54: 249-262.

Chisolm, J. J., Jr.; Mellits, E. D.; Quaskey, S. A. (1985) The relationship between the level of lead absorption in children and the age, type and condition of housing. Environ. Res. 38: 31-45.

Christoffersson, J. O.; Schutz, A.; Ahlgren, L.; Haeger-Aronsen, B.; Mattsson, S.; Skerfvig, S. (1984) Lead in finger-bone analyzed *in vivo* in active and retired lead workers. Am. J. Ind. Med. 6: 447-457.

Ciriello, P. L.; Goldberg, T. (1987) Draft report: lead-contaminated soil cleanup. Boston, MA: U.S. Environmental Protection Agency, Region I, March 27, 1987. (Appears in this report as Appendix E).

City of Dallas Department of Health. (1985) Blood lead screening program: East Oak Cliff and West Dallas. Final report: Fall.

City of Chicago, IL. (1985) Department of Health lead screening statistics: 1985 report.

Clark, C. S.; Bornschein, R. L.; Succop, P.; Que Hee, S. S.; Hammond, P. B.; Peace, B. (1985) Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ. Res. 38: 46-53.

Clark, C. S.; Bornschein, R. L.; Succop, P.; Hammond, P. B.; Peace, B.; Krafft, K.; Dietrich, K. (1987) Pathways to elevated blood lead and their importance in control strategy development. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 159-161.

Dacre, J. C.; Ter Haar, G. L. (1977) Lead levels in tissues from rats fed soils containing lead. Arch. Environ. Contam. Toxicol. 6: 111-119.

Davis, J. M.; Svendsgaard, D. J. (1987) Low-level lead exposure and child development. Nature (London) 329: 297-300.

de la Burde, B.; Choate, M. S., Jr. (1972) Does asymptomatic lead exposure in children have latent sequelae? J. Pediatr. 87: 1088-1091.

de la Burde, B.; Choate, M. S., Jr. (1975) Early asymptomatic lead exposure and development at school age. J. Pediatr. 87: 638-642.

Delves, H. T.; Clayton, B. E.; Carmichael, A.; Bubear, M.; Smith, M. (1982) An appraisal of the analytical significance of tooth-lead measurements as possible indices of environmental exposure of children to lead. Ann. Clin. Biochem. 19: 329-337.

DeSilva, P. E. (1981) Determination of lead in plasma and studies on its relationship to lead in erythrocytes. Br. J. Ind. Med. 38: 209-217.

Dietrich, K. N.; Krafft, K. M.; Bier, M.; Succop, P. A.; Berger, O.; Bornschein, R. L. (1986) Early effects of fetal lead exposure: neurobehavioral findings at 6 months. Int. J. Biosoc. Res. 8: 151-168.

Dietrich, K. N.; Krafft, K. M.; Shukla, R.; Bornschein, R. L.; Succop, P. A. (1987a) The neurobehavioral effects of early lead exposure. In: Schroeder, S. R., ed. Toxic substances and mental retardation: neurobehavioral toxicology and teratology. Washington, DC: American Association on Mental Deficiency; pp. 71-95. (Begab, M. J., ed. Monographs of the American Association on Mental Deficiency: no. 8).

Dietrich, K. N.; Krafft, K. M.; Bier, M.; Berger, O.; Succop, P. A.; Bornschein, R. L. (1987b) Neurobehavioral effects of fetal lead exposure: the first year of life. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Dietrich, K. N.; Krafft, K. M.; Bornschein, R. L.; Hammond, P. B.; Berger, O.; Succop, P. A.; Bier, M. (1987c) Effects of low-level fetal lead exposure on neurobehavioral development in early infancy. Pediatrics: in press.

Duggan, M. J.; Inskip, M. J. (1985) Childhood exposure to lead in surface dust and soil: a community health problem. Public Health Rev. 13: 1-54.

Elinder, C.-G.; Gerhardsson, L.; Oberdöster, G. (1987) Biological monitoring of toxic metals: final report of a conference, biological monitoring of metals, June 1986; Rochester, NY: University of Rochester. (in press).

Ernhart, C. B.; Landa, B.; Schell, N. B. (1981) Subclinical levels of lead and developmental deficit--a multivariate follow-up reassessment. Pediatrics 67: 911-919.

Ernhart, C. B.; Wolf, A. W.; Kennard, M. J.; Filipovich, H. F.; Sokol, R. J.; Erhard, P. (1985a) Intrauterine lead exposure and the status of the neonate. In: Lekkas, T. D., ed. International conference: heavy metals in the environment; September. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 35-37.

Ernhart, C. B.; Landa, B.; Wolf, A. W. (1985b) Subclinical lead levels and development deficit: reanalysis of data. J. Learning Disabilities 18: 475-479.

Ernhart, C. B.; Wolf, A. W.; Kennard, M. J.; Erhard, P.; Filipovich, H. F.; Sokol, R. J. (1986) Intrauterine exposure to low levels of lead: the status of the neonate. Arch. Environ. Health 41: 287-291.

Ernhart, C. B.; Morrow-Tlucak, M.; Marler, M. R.; Wolf, A. W. (1987a) Low level lead exposure in the prenatal and early preschool periods: early preschool development. Neurotoxicol. Teratol. 9: 259-270.

Ernhart, C. B.; Brittenham, G.; Marler, M. R.; Sokol, R. J. (1987b) Lead related birth defects: some methodological issues. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Everson, J.; Patterson, C. C. (1980) "Ultra-clean" isotope dilution/mass spectrometric analyses for lead in human blood plasma indicate that most reported values are artificially high. Clin. Chem. 26: 1603-1607.

Facchetti, S. (1985) Isotope lead experiment--an update. Presented at: Lead environmental health: the current issues; April/May; Durham, NC: Duke University.

Facchetti, S.; Geiss, F. (1982) Isotopic lead experiment: status report. Luxembourg: Commission of the European Communities; publication no. EUR 8352EN.

Farfel, M. R. (1985) Reducing lead exposure in children. Annu. Rev. Public Health 6: 333-360.

Farfel, M. R.; Chisolm, J. J., Jr. (1987) Comparison of traditional and alternative residential lead paint removal methods. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 2; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 212-214.

Flanagan, P. R.; Chamberlain, M. J.; Valberg, L. S. (1982) The relationship between iron and lead absorption in humans. Am. J. Clin. Nutr. 36: 823-829.

Friberg, L. T. (1985) Yant memorial lecture. The rationale of biological monitoring of chemicals--with special reference to metals. Am. Ind. Hyg. Assoc. J. 46: 633-642.

Friberg, L. T.; Vahter, M. (1983) Assessment of exposure to lead and cadmium through biological monitoring: results of a UNEP/WHO global study. Environ. Res. 30: 95-128.

Fulton, M.; Thomson, G.; Hunter, R.; Raab, G.; Laxen, D.; Hepburn, W. (1987) Influence of blood lead on the ability and attainment of children in Edinburgh. Lancet (1): 1221-1225.

Galke, W. A.; Hammer, D. I.; Keil, J. E., Lawrence, S. W. (1975) Environmental determinants of lead burdens in children. In: Hutchinson, T. C.; Lestein, S.; Page, A. L.; Van Loon, F.; Davey, T. eds. International conference on heavy metals in the environment, v. 3; October; Toronto, Ontario, Canada; pp. 53-74.

GCA Corporation. (1985) Estimated numbers of children residing near lead point sources. Research Triangle Park, NC: Office of Air Quality Planning and Standards, Ambient Standards Branch; contract no. 68-02-3804.

338

Gibson, J. L. (1904) A plea for painted railings and painted walls of rooms as the source of lead poisoning among Queensland children. Australia Med. Gazette 23: 149-153.

Gibson, J. L.; Love, W.; Hardine, D.; Bancroft, P.; Turner, A. J. (1892) Note on lead poisoning as observed among children in Brisbane. In: Huxtable, L. R., ed. Trans. 3rd Intercolonial Medical Congress of Australasia; September. Sydney, Australia: Charles Potter; pp. 76-83.

Gilsinn, J. (1972). Estimate of the nature and extent of lead paint poisoning in the United States. Washington, DC: National Bureau of Standards; NBS technical note 764.

Grant, L. D.; Davis, J. M. (1987) Effect of low-level lead exposure on pediatric neurobehavioral and physical development: current findings and future directions. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Graziano, J.; Popovac, M.; Murphy, A.; Mehmeti, A.; Kline, J.; Ahmedi, G.; Shrout, P.; Zvicer, Z.; Wasserman, G.; Gashi, E.; Stein, B.; Rajovic, B.; Belmong, L.; Colakovic, B.; Bozovic, R.; Haxhiu, R.; Radovic, L.; Vlaskovic, R.; Loiacano, N. (1987) Environmental lead, reproduction and infant development. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Griffin, T. B.; Coulston, F.; Goldberg, L.; Wills, H.; Russell, J. C.; Knelson, J. H. (1975) Clinical studies on men continuously exposed to airborne particulate lead: In: Griffin, T. B.; Knelson, J. H., eds. Lead. v. 2, supplement. Stuttgart, West Germany: Georg Thieme Publishers; pp. 221-240.

Gross, S. B. (1981) Human oral and inhalation exposures to lead: summary of Kehoe balance experiments. J. Toxicol. Environ. Health 8: 333-377.

Hamilton, A.; Reznikoff, P.; Burnham, G. M. (1925) Tetraethyl lead. JAMA J. Am. Med. Assoc. 84: 1481-1486.

Hammond, P. B. (1973) The effects of D-penicillamine on the tissue distribution and excretion of lead. Toxicol. Appl. Pharmacol. 26: 241-246.

Harley, N. H.; Kneip, T. H. (1985) An integrated metabolic model for lead in humans of all ages: final report. New York, NY: New York University School of Medicine; EPA contract no. B44899.

Harvard School of Public Health Community Health Improvement Program. (1981) Casas Sin Plomo: final report. Cambridge, MA: Harvard University.

Harvey, P. G.; Hamlin, M. W.; Kumar, R.; Delves, H. T. (1984) Blood lead, behavior and intelligence test performance in pre-school children. Sci. Total Environ. 40: 45-60.

329

Hatzakis, A.; Kokkevi, A.; Katsouvanni, K.; Maravelias, K.; Salaminios, F.; Kalandidi, A.; Koutselinis, A.; Stefanis, K.; Trichopoulos, D. (1987) Lead exposure and children's cognitive functions and behavior. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 204-209.

Heard, M. J.; Chamberlain, A. C. (1982) Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. Hum. Toxicol. 1: 411-415.

Heard M. J.; Chamberlain, A. C. (1983) Uptake of lead by humans and effects of minerals and food. Sci. Total Environ. 30: 245-253.

Hofmann, W.; Steinhausler, H.; Pohl, E. (1979) Dose calculation for the respiratory tract from inhaled natural radioactive nuclides as a function of age. I. Compartmental deposition, retention and resulting dose. Health Phys. 37: 517-532.

Hryhorczuk, D. O.; Rabinowitz, M. B.; Hessl, S. M.; Hoffman, D.; Hogan, M. M.; Mallin, K.; French, H.; Arris, P.; Berman, E. (1985) Elimination kinetics of blood lead in workers with chronic lead intoxication. Am. J. Ind. Med. 8: 33-42.

Hunter, J.; Urbanowicz, M. A.; Yule, W.; Lansdown, R. (1985) Automated testing of reaction time and its association with lead in children. Int. Arch. Occup. Environ. Health 57: 27-34.

James, A. C. (1978) Lung deposition of sub-micron aerosols calculated as a function of age and breathing rate. In: National Radiological Protection Board annual research and development report. Harwell, United Kingdom: National Radiological Protection Board, Atomic Energy Research Establishment; pp. 71-75.

Jelinek, C. F. (1982) Levels in lead in the United States food supply. J. Assoc. Off. Anal. Chem. 65: 942-946.

Johnson, J.; Paul, R. (1986) Estimation of daily lead uptake in children and resulting end-of-month blood lead levels. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards; contract no. 68-02-4309.

Johnson, N. E.; Tenuta, K. (1979) Diets and lead blood levels of children who practice pica. Environ. Res. 18: 369-376.

Kang, H. K.; Infante, P. F.; Carra, J. S. (1983) Determination of blood-lead elimination patterns of primary lead smelter workers. J. Toxicol. Environ. Health 11: 199-210.

Kjellstrom, T. (1985) In: Friberg, L.; Elinder, C. G.; Kjellstrom, T.; Nordberg, G. S., eds. Critical organs, critical concentrations and whole-body dose-response relationships, in cadimum and health. A toxicological and epidemiological appraisal: v. 2. Boca Raton, FL: CRC Press.

Klein, H.; Namer, R.; Harper, E.; Corbin, R. (1970) Earthenware containers as a source of fatal lead poisoning. N. Engl. J. Med. 283: 669-672.

Kneip, T. J.; Mallon, R. P.; Harley, N. H. (1983) Biokinetic modeling for mammalian lead metabolism. Neurotoxicology 4: 189-192.

Kotok, D.; Kotok, R.; Heriot, T. (1977) Cognitive evaluation of children with elevated blood lead levels. Am. J. Dis. Child. 131: 791-793.

Lansdown, R.; Yule, W.; Urbanowicz, M.-A.; Hunter, J. (1986) The relationship between blood-lead concentrations, intelligence, attainment and behaviour in a school population: the second London study. Int. Arch. Occup. Environ. Health 57: 225-235.

Lauwers, M. C.; Hauspie, R. C.; Susanne, C.; Verheyden, J. (1986) Comparison of biometric data of children with high and low levels of lead in the blood. Am. J. Phys. Anthropol. 69: 107-116.

Lerman, D. L. (1986) How well can alternate policies reduce rural substandard housing? Washington, DC: U.S. Department of Agriculture, Economic Research Service; Rural Development Research Service report no. 67.

Levin, R. (1987) Lead in U.S. public drinking water: the benefits of reducing that exposure. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 2; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 215-217.

Lin-Fu, J. S. (1982a) Children and lead: new findings and concerns. N. Engl. J. Med. 307: 615.

Lin-Fu, J. S. (1982b) The evolution of childhood lead poisoning as a public health program. In: Chisolm, J. J., Jr.; O'Hara, D. M., eds. Lead absorption in children: management, clinical and environmental aspects. Baltimore, MD: Urban and Schwartzenberg; pp. 1-10.

Lin-Fu, J. S. (1985a) Undue lead absorption in children: scope of the problem--the nation and New England: proceedings of symposium on clinical management of children with undue lead absorption; April; Worcester, MA. Worcester, MA: New England Consortium of Childhood Lead Paint Poisoning Programs.

Lin-Fu, J. S. (1985b) Childhood lead poisoning: the national perspective: symposium for health care providers--childhood lead poisoning: is it still a problem? Cincinnati, OH: Children's Hospital Research Foundation.

Lin-Fu, J. S. (1987) Review of second draft of ATSDR Report to Congress. Memorandum: Bureau of Health Care Delivery and Assistance, HHS, to Agency for Toxic Substances and Disease Registry. April 8, 1987.

Lippmann, M. (1986) [Letter regarding review of Air Quality Criteria for Lead]. Washington, D.C.: U.S. Environmental Protection Agency, Clean Air Scientific Committee; August 29.

Mahaffey, K. R. (1982) Role of nutrition in prevention of pediatric lead toxicity. In: Chisolm, J. J., Jr.; O'Hara, D. M. eds. Lead absorption in children: management, clinical and environmental aspects. Baltimore, MD: Urban and Schwartzenberg; pp. 63-78.

Mahaffey, K. R.; Annest, J. L. (1986) Association of erythrocyte protoporphyrin with blood lead level and iron status in the second National Heath and Nutrition Examination Survey, 1976-1980. Environ. Res. 41: 327-338.

Mahaffey, K. R.; Michaelson, I. A. (1980) The interaction between lead and nutrition. In: Needleman, H. L., ed. Low level lead exposure: the clinical implications of current research. New York, NY: Raven Press; pp. 159-200.

Mahaffey, K. R.; Treloar, S.; Banks, T. A. (1976) Differences in dietary intake of calcium, phosphorus and iron of children having normal and elevated blood lead concentrations. J. Nutr. 106(7):xxx.

Mahaffey, K. R.; Annest, J. L.; Roberts, J.; Murphy, R. S. (1982) National estimates of blood lead levels: United States 1976-1980: association with selected demographic and socioeconomic factors. N. Engl. J. Med. 307: 573-579.

Mahaffey, K. R.; Gartside, P. S.; Glueck, C. J. (1986) Blood lead levels and dietary calcium intake in 1- to 11-year old children: the Second National Health and Nutrition Examination Survey, 1976-1980. Pediatrics 78: 257-262.

Manton, W. I. (1985) Total contribution of airborne lead to blood lead. Br. J. Ind. Med. 42: 168-172.

Markowitz, M. E.; Rosen, J. F. (1981) Zinc (Zn) and copper (Cu) metabolism in CaNa$_2$ EDTA-treated children with plumbism. Pediatr. Res. 15: 635.

McBride, W. G.; Black, B. P.; English, B. J. (1982) Blood lead levels and behavior of 400 pre-school children. Med. J. Aust. 2: 26-29.

McBride, W. G.; Cooney, G. C.; Bell, A. (1987) Blood lead levels in Sydney urban children. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 153-155.

McKhann, C. F. (1926) Lead poisoning in children. Am. J. Dis. Child. 2: 386-392.

McMichael, A. J.; Vimpani, G. V.; Robertson, E. F.; Baghurst, P. A.; Clark, P. D. (1986) The Port Pirie cohort study: maternal blood lead and pregnancy outcome. J. Epidemiol. Commun. Health 40: 18-25.

Mielke, H.; Blake, B.; Burroughs, S.; Hassinger, N. (1984) Urban lead levels in Minneapolis: the case of the Hmong children. Environ. Res. 34: 64-76.

Milar, C. R.; Mushak, P. (1982) Lead-contaminated house dust: hazard, measurement and decontamination. In: Chisolm, J. J., Jr.; O'Hara, D. M., eds. Lead absorption in children: management, clinical and environmental aspects, Baltimore, MD: Urban and Schwartzenberg; pp. 143-152.

Miller, T. R.; Toulmin, L. (1987) Economic analyses of lead paint regulations governing HUD-assisted and FHA insured housing: final report. Washington, DC: U.S. Department of Housing and Urban Development; Urban Institute Project 3673-01.

Moore, M. R.; Meredith, P. A.; Watson, W. S.; Sumner, D. J.; Taylor, M. K.; Goldberg, A. (1980) The percutaneous absorption of lead-203 in humans from cosmetic preparations containing lead acetate, as assessed by whole-body counting and other techniques. Food Cosmet. Toxicol. 18: 399-405.

Moore, M. R.; Goldberg, A.; Pocock, S. J.; Meredith, P. A.; Stewart, I. M.; Macanespie, H.; Lees, R.; Low, A. (1982) Some studies of maternal and infant lead exposure in Glasgow. Scott. Med. J. 27: 113-122.

Moore, M. R.; Bushnell, I. W. R.; Goldberg, A. (1987) A prospective study of the results of changes in environmental lead exposure in children in Glasgow. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Morrow, P. E.; Beiter, H.; Amato, F.; Gibb, F. R. (1980) Pulmonary retention of lead: an experimental study in man. Environ. Res. 21: 373-384.

Morse, D. L.; Watson, W. N.; Housworth, J.; Witherell, L. E.; Landrigan, P. J. (1979) Exposure of children to lead in drinking water. Am. J. Public Health 69: 711-712.

Mushak, P. (1987) Biological monitoring of lead exposure in children: overview of selected biokinetic and toxicological issues. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Mushak, P.; Schroeder, C. (1981) Multiple media pollutant exposures and their regulation. Washington, DC: National Commission on Air Quality; contract no. 23 AQ 6981.

National Food Processors Association. (1986) Retail market survey data for lead in canned foods. Washington, DC: August 7.

National Research Council. (1972) Airborne lead in perspective. Washington, DC: Committee on Medical and Biologic Effects of Atmospheric Pollutants.

National Research Council. (1976) Recommendations for the prevention of lead poisoning in children. Washington, DC: National Academy of Sciences.

National Research Council. (1980) Lead in the human environment. Washington, DC: National Academy of Sciences.

National Research Council. (1987) Risking the future. Adolescent sexuality, pregnancy, and childbearing: v. 1. Cheryl B. Hayes, ed. Washington, DC: National Academy Press.

Needleman, H. L., ed. (1980) Low level lead exposure: clinical implications of current research. New York, NY: Raven Press.

Needleman, H. L. (1987) Low level lead exposure and children's intelligence: a quantitative and critical review of modern studies. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 1-8.

Needleman, H. L.; Bellinger, D. C. (1987) Type II fallacies in the study of childhood exposure to lead at low dose: a critical and quantitative review. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Needleman, H. L.; Shapiro, I. M. (1974) Dentine lead levels in asymptomatic Philadelphia school children: subclinical exposure in high and low risk groups. EHP Environ. Health Perspect. 7: 27-31.

Needleman, H. L.; Gunnoe, C.; Leviton, A.; Reed, R.; Peresie, H.; Maher, C.; Barrett, P. (1979) Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N. Engl. J. Med. 300: 689-695.

Needleman, H. L.; Leviton, A.; Bellinger, D. (1982) Lead-associated intellectual deficit [letter]. N. Engl. J. Med. 306: 367.

Needleman, H. L.; Rabinowitz, M.; Leviton, A.; Linn, S.; Schoenbaum, E. (1984) The relationship between prenatal exposure to lead and congenital anomalies. JAMA J. Am. Med. Assoc. 251: 2956-2959.

New York City, NY. (1982) Administrative Code 26-12.08, January, 1982.

New York City Department of Health. (1985) Bureau of Lead Poisoning Control: annual report.

O'Flaherty, E. J.; Hammond, P. B.; Lerner, S. I. (1982) Dependence of apparent blood lead half-life on the length of previous lead exposure in humans. Fundam. Appl. Toxicol. 2: 49-54.

O'Hara, D. M. (1982) Social factors in the recurrence of increased lead absorption. In: Chisolm, J. J., Jr.; O'Hara, D. M., eds. Lead absorption in children. Management, clinical and environmental aspects. Baltimore, MD: Urban and Schwartzenberg; pp. 89-101.

Otto, D. A.; Benignus, V. A.; Muller, K. E.; Barton, C. N. (1981) Effects of age and body lead burden on CNS function in young children. I. Slow cortical potentials in young children. Electroencephalogr. Clin. Neurophysiol. 52: 229-239.

Otto, D. A.; Benignus, V. A.; Muller, K. E.; Barton, C.; Seiple, K.; Prah, J.; Schroeder, S. R. (1982) Effects of low to moderate lead exposure on slow cortical potentials in young children: two year follow-up study. Neurobehav. Toxicol. Teratol. 4: 733-737.

Otto, D. A.; Robinson, G.; Baumann, S.; Schroeder, S. R.; Mushak, P.; Kleinbaum, D.; Boone, L. (1985) Five-year follow-up study of children with low-to-moderate lead absorption: electrophysiological evaluation. Environ. Res. 38: 168-186.

Patterson, C. C. (1965) Contaminated and natural lead environments of man. Arch. Environ. Health 11: 344-360.

Pennington, J. A. T. (1983) Revision of the total diet study food list and diets. J. Am. Diet. Assoc. 82: 166-173.

Perino, J.; Ernhart, C. B. (1974) The relation of subclinical lead level to cognitive and sensory-motor impairment in black pre-schoolers. J. Learn. Dis. 7: 616-620.

Perlstein, M. A.; Attala, R. (1966) Neurologic sequelae of plumbism in children. Clin. Pediatr. 5: 292-298.

Piomelli, S.; Corash, L.; Corash, M. B.; Seamon, C.; Mushak, P.; Glover, B.; Padgett, R. (1980) Blood lead concentrations in a remote Himalayan population. Science (Washington, DC) 210: 1135-1137.

Piomelli, S.; Seaman, C.; Zullow, D.; Curran, A.; Davidow, B. (1982) Threshold for lead damage to heme synthesis in urban children. Proc. Natl. Acad. Sci. U. S. A. 79: 3335-3339.

Piomelli, S.; Rosen, J. F.; Chisolm, J. J., Jr.; Graef, J. W. (1984) Management of childhood lead poisoning. J. Pediatr. 105: 523-531.

Pocock, S. J.; Ashby, D.; Smith, M. A. (1987) Lead exposure and children's intellectual performance. Int. J. Epidemiol. 16: 57-67.

Pope, A. (1986) Exposure of children to lead-based paints. Research Triangle Park, NC: U.S. Environmental Protection Agency, Strategies and Air Standards Division; EPA contract no. 68-02-4329.

Public Health Foundation. (1986) Special report: State Health Agency lead poisoning prevention activities, 1983. Washington, DC: Public Health Foundation.

Que Hee, S. S.; Peace, B.; Clark, C. S.; Boyle, J. R.; Bornschein, R. L.; Hammond, P. B. (1985) Evolution of efficient methods to sample lead sources, such as house dust and hand dust, in the homes of children. Environ. Res. 38: 77-95.

Rabinowitz, M. B.; Needleman, H. L. (1982) Temporal trends in the lead concentrations of umbilical cord blood. Science (Washington, DC) 216: 1429-1431.

Rabinowitz, M. B.; Wetherill, G. W.; Kopple, J. D. (1976) Kinetic analysis of lead metabolism in healthy humans. J. Clin. Invest. 58: 260-270.

Rabinowitz, M. B.; Wetherill, G. W.; Kopple, J. D. (1977) Magnitude of lead intake from respiration by normal man. J. Lab. Clin. Med. 90: 238-248.

Rabinowitz, M. B.; Kopple, J. D.; Wetherill, G. W. (1980) Effect of food intake and fasting on gastrointestinal lead absorption in humans. Am. J. Clin. Nutr. 33: 1784-1788.

Rabinowitz, M.; Leviton, A.; Bellinger, D. C. (1985) Home refinishing: lead paint and infant blood lead levels. Am. J. Public Health 75: 403-404.

Rey-Alvarez, S.; Menke-Hargrove, T. (1987) Deleading dilemma: pitfall in the management of childhood lead poisoning. Pediatrics 79: 214-217.

Robinson, G. S.; Baumann, S.; Kleinbaum, D.; Barton, C.; Schroeder, S. R.; Mushak, P.; Otto, D. A. (1985) Effects of low to moderate lead exposure on brainstem auditory evoked potentials in children. Copenhagen, Denmark: WHO Regional Office for Europe; pp. 177-182. (Environmental health document 3).

Robinson, G. S.; Keith, R. W.; Bornschein, R. L.; Otto, D. A. (1987) Effects of environmental lead exposure on the developing auditory system as indexed by the brainstem auditory evoked potential and pure tone hearing evaluations in young children. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 223-225.

Roels, H. A.; Buchet, J.-P.; Lauwerys, R. R.; Bruaux, P.; Claeys-Thoreau, F.; Lafontaine, A.; Verduyn, G. (1980) Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. Environ. Res. 22: 81-94.

Rosen, J. F. (1983) The metabolism in isolated bone cell populations: interactions between lead and calcium. Toxicol. Appl. Pharmacol. 71: 101-112.

Rosen, J. F. (1985) Metabolic and cellular effects of lead: a guide to low level lead toxicity in children. In: Mahaffey, K. R., ed. Dietary and environmental lead: human health effects. Amsterdam, The Netherlands: Elsevier; pp. 157-185.

Rosner, D.; Markowitz, G. A. (1985) 'Gift of God?' The public health controversy over leaded gasoline during the 1920s. Am. J. Public Health 75: 344-352.

Rothenberg, S. J.; Schnaas, L.; Neri-Mendez, C. Z. (1987) The pilot study of the Mexico City prospective lead study: neurobehavioral newborn status and prenatal and perinatal blood lead levels. In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Rummo, J. H. (1974) Intellectual and behavioral effects of lead poisoning in children [Ph.D. thesis]. Chapel Hill, NC: University of North Carolina. Available from: University Microfilms, Ann Arbor, MI; publication no. 74-26930.

Rummo, J. H.; Routh, D. K.; Rummo, N. J.; Brown, J. F. (1979) Behavioral and neurological effects of symptomatic and asymptomatic lead exposure in children. Arch. Environ. Health 34: 120-124.

Ryu, J. E.; Ziegler, E. E.; Nelson, S. E.; Fomen, S. J. (1983) Dietary intake of lead and blood lead concentration in early infancy. Am. J. Dis. Child. 137: 886-891.

Ryu, J. E.; Ziegler, E. E.; Nelson, S. E.; Fomen S. J. (1985) Dietary and environmental exposure to lead and blood during early infancy. In: Mahaffey, K. R., ed. Dietary and environmental lead: human health effects. Amsterdam, The Netherlands: Elsevier Press; pp. 187-209.

Saenger, P.; Rosen, J. F.; Markowitz, M. E. (1982) Diagnostic significance of edetate disodium calcium testing in children with increased lead absorption. Am. J. Dis. Child. 136: 312-315.

Saenger, P.; Markowitz, M. E; Rosen, J. F. (1984) Depressed excretion of 6Beta-hydroxycortisol in lead-toxic children. J. Clin. Endocrinol. Metab. 58: 363-367.

Schier, D.; Hall, W. (1977) Analysis of housing data collected in a lead-based paint survey in Pittsburgh, Pa. Part I. Washington, DC: National Bureau of Standards; report no. NBSIR 77-1250.

Schneider, D. J.; Lavenhar, M. A. (1986) Lead poisoning: more than a medical problem. Am. J. Public Health 76: 242-244.

Schroeder, S. R.; Hawk, B. (1987) Child-caregiver environmental factors related to lead exposure and IQ. In: Schroeder, S. R., ed. Toxic substances and mental retardation: neurobehavioral toxicology and teratology. Washington, DC: American Association on Mental Deficiency; (Begab, M. J., ed. Monographs of the American Association on Mental Deficiency: no. 8); pp. 97-137.

Schroeder, S. R.; Hawk, B.; Otto, D. A.; Mushak, P.; Hicks, R. E. (1985) Separating the effect of lead and social factors on IQ. Environ. Res. 38: 144-154.

Schwartz, J.; Otto, D. A. (1987) Blood lead, hearing thresholds, and neurobehavioral development in children and youth. Arch. Environ. Health. 42: 153-160.

Schwartz, J.; Pitcher, H.; Levin, R.; Ostro, B.; Nichols, A. L. (1985) Costs and benefits of reducing lead in gasoline: final regulatory impact analysis. Washington, DC: U. S. Environmental Protection Agency, Office of Policy, Planning and Evaluation; EPA report no. EPA-230/05-85-006.

Schwartz, J.; Angle, C.; Pitcher, H. (1986) Relationship between childhood blood lead levels and stature. Pediatrics 77: 281-288.

Shukla, R.; Bornschein, R. L.; Dietrich, K. N.; Mitchell, T.; Grote, J.; Berger, O.; Hammond, P. B.; Succop, P. A. (1987) Effects of fetal and early postnatal lead exposure on child's growth in stature - the Cincinnati lead study. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 210-212.

Silbergeld, E. K. (1983a) Localization of metals: issues of importance to neurotoxicology of lead. Neurotoxicology 4: 193-200.

Silbergeld, E. K. (1983b) Experimental studies of lead neurotoxicity: implication for mechanisms, dose-response and reversibility, In: Rutter, M.; Russell Jones, R., eds. Lead versus health. London, United Kingdom: John Wiley and Sons, Ltd.; pp. 191-218.

Silbergeld, E. K.; Wolinsky, J. S.; Goldstein, G. W. (1980) Electron probe microanalysis of isolated brain capillaries poisoned with lead. Brain Res. 189: 369-376.

Smith, M. (1985) Recent work on low level lead exposure and its impact on behavior, intelligence, and learning: a review. J. Am. Acad. Child Psychiatry 24: 24-32.

Smith, M.; Delves, H. T.; Lansdown, R.; Clayton, B.; Graham, P. (1983) The effects of lead exposure on urban children: the Institute of Child Health/Southhampton study. Dev. Med. Child Neurol. 25 (suppl. 47).

Sorrell, M.; Rosen, J. F.; Roginsky, M. (1977) Interactions of lead, calcium, vitamin D and nutrition in lead-burdened children. Arch. Environ. Health 32: 160-164.

Steenhout, A.; Pourtois, M. (1981) Lead accumulation in teeth as a function of age with different exposures. Br. J. Ind. Med. 38: 297-303.

Succop, P. A.; O'Flaherty, E. J.; Bornschein, R. L.; Clark, C. S.; Krafft, K.; Hammond, P. B.; Shukla, R. (1987) A kinetic model for estimating changes in the concentration of lead in the blood of young children. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 2; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 289-291.

Tola, S.; Hernberg, S.; Asp, S.; Nikkanen, J. (1973) Parameters indicative of absorption and biological effect in new lead exposure: a prospective study. Br. J. Ind. Med. 30: 134-141.

TRC Environmental Consultants, Inc. (1986) Exposure to airborne lead from stationary sources: an evaluation of proposed national ambient air quality standards for lead. Wethersfield, CT: TRC Environmental Consultants, Inc.; project no. 3220-551.

Ulvund, S. E. (1984) Predictive validity of assessments of early cognitive competence in light of some current issues in developmental psychology. Hum. Development 27: 76-83.

U.S. Bureau of the Census. (1983) Census of population, 1980. Characteristics of the population, general social and economic statistics: United States summary. Washington, DC: U.S. Department of Commerce.

U.S. Bureau of the Census. (1984) Current population reports, series P-25, no. 952, projections of the population of the United States by age, sex and race: 1983 to 2080. Washington, DC: U.S. Department of Commerce.

U.S. Bureau of the Census. (1986) American housing survey, 1983. Part B: indicators of housing and neighborhood quality by financial characteristics, December. Washington, DC: U.S. Department of Commerce.

U.S. Department of Health and Human Services, Public Health Service, National Center for Heath Statistics. (1984) Vital statistics of the United States, 1980, V. I, natality; Hyattsville, MD; Table 1-59.

U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics. (1985a) Vital Statistics of the United States, 1981: V. I, natality; Hyattsville, MD; 1985, table 1-59.

U.S. Department of Health and Human Services, Public Health Service National Center for Health Statistics (1985b). Vital Statistics of the Untied States, 1980, V. II, mortality part B; Hyattsville, MD; table 8-1.

U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics. (1986) Vital statistics of the United States, 1981, V. II, mortality part B; Hyattsville, MD; table 8-1.

U.S. Environmental Protection Agency. (1977) Air quality criteria for lead. Research Triangle Park, NC: Health Effects Research Laboratory; EPA report no. EPA-600/8-77-017 Available from: NTIS, Springfield, VA; PB-280411.

U.S. Environmental Protection Agency. (1984) Costs and benefits of reducing lead in gasoline (interim draft: J. Schwartz, J. Leggett, B. Ostro, H. Pitcher, R. Levin, authors). Washington, DC: Office of Policy Analysis.

U.S. Environmental Protection Agency. (1985) Costs and benefits of reducing lead in gasoline: final regulatory impact analysis. Washington, DC: Office of Policy, Planning and Evaluation (Schwartz, J.; Pitcher, H.; Levin, R.; Ostro, B.; Nichols, A. L.); EPA report no. EPA-230/05-85-006.

U.S. Enviromental Protection Agency. (1986a) Air quality criteria for lead. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office: EPA report no. EPA-600/8-83/028aF-dF. 4v. Available from: NTIS, Springfield, VA; PB87-142378.

U.S. Environmental Protection Agency. (1986b) Reducing lead in drinking water: a benefit analysis. Washington, DC: Office of Policy Planning and Evaluation; EPA report no. EPA-230-09-86-019.

U.S. Environmental Protection Agency. (1986c) Review of the national ambient air quality standards for lead. Assessment of scientific and technical information: draft staff paper. Research Triangle Park, NC: Office of Air Quality Planning and Standards.

U.S. Environmental Protection Agency. (1987a) Letters from Jeanne Briskin, Office of Drinking Water to Frank L. Mitchell, D. O., ATSDR.

U.S. Environmental Protection Agency. (1987b) [Letter from Jeanne Briskin]. Research Triangle Park, NC: Environmental Criteria and Assessment Office; October 9.

U.S. Environmental Protection Agency. (1987c) Preliminary results: the aging solder study. Washington, DC: Office of Drinking Water.

U.S. Navy. (1987) Data on results of analyses of potable tap water samples. Obtained from Mr. Chuck Roydhouse, Safety Officer, David Taylor Naval Ship research and Development Center, Annapolis, MD 21402.

Vimpani, G. V.; Wigg, N. R.; Robertson, E. F.; McMichael, A. J.; Baghurst, P. A.; Roberts, R. R. (1985) The Port Pirie cohort study: blood lead concentration and childhood development assessment. Presented at: Lead environmental health: the current issues; May; Durham, NC: Duke University.

Vimpani, G. V.; Baghurst, P. A.; Wigg, N. R.; Robertson, E. F.; McMichael, A. J.; Roberts, R. R. (1987) The Port Pirie cohort study - cumulative lead exposure and neurodevelopmental status at age 2 years: do HOME scores and maternal IQ reduce apparent effects of lead on Bayley Mental scores? In: Smith, M.; Grant, L. D.; Sors, A., eds. Lead exposure and child development: an international assessment. Lancaster, United Kingdom: MTP Press; in press.

Wallace, J. E. (1986) The cost of lead based paint abatement in public housing: final report. Contract HC-5685, Abt. Associates, Inc. For the Office of Policy Development and Research, U.S. Department of Housing and Urban Development, July, 1986.

Walter, S. D.; Yanke, A. J.; von Lindern, I. H. (1980) Age-specific risk factors for lead absorption in children. Arch. Environ. Health 35: 53-58.

Watson, W. S.; Hume, R.; Moore, M. R. (1980) Oral absorption of lead and iron. Lancet (8188): 236-237.

Wedeen, R. P. (1984) Poison in the pot: the legacy of lead. Carbondale, IL: Southern Illinois University Press.

Winneke, G.; Hrdina, K.-G.; Brockhaus, A. (1982) Neuropsychological studies in children with elevated tooth lead concentrations. Part I: Pilot study. Int. Arch. Occup. Environ. Health 51: 169-183.

Winneke, G.; Kramer, U.; Brockhaus, A.; Ewers, U.; Kujanek, G.; Lechner, H.; Janke, W. (1983) Neuropsychological studies in children with elevated tooth-lead concentration. Part II. Extended study. Int. Arch. Occup. Environ. Health 51: 231-252.

Winneke, G.; Beginn, U.; Ewert, T.; Havestadt, C.; Kramer, U.; Krause, C.; Thron, H. L.; Wagner, H. M. (1984) [Study of the measurement of subclinical lead effects on the nervous system of Nordenham children with known pre-natal exposure]. Schriftenr. Verwasser. Boden. Lufthyg. (59): 215-229.

Winneke, G.; Collet, W.; Kramer, U.; Brockhaus, A.; Ewert, T.; Krause, C. (1987a) Three- and six-year follow-up studies in lead exposed children. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 60-62.

Winneke, G.; Munoz, C.; Lilienthal, H. (1987b) Neurobehavioral effects of lead: the reversibility issue. In: Lindberg, S. E.; Hutchinson, T. C., eds. International conference: heavy metals in the environment, v. 1; September; New Orleans, LA. Edinburgh, United Kingdom: CEP Consultants, Ltd.; p. 66.

Wolnik, K. A.; Fricke, F. L.; Capar, S. G.; Braude, G. L.; Meyer, M. W.; Satzger, R. D.; Bonner, E. (1983) Elements in major raw agricultural crops in the United States. I. Cadmium and lead in lettuce, peanuts, potatoes, soybeans, sweet corn and wheat. J. Agr. Food Chem. 31: 1240-1244.

Wolf, A. W.; Ernhart, C. B.; White, C. S. (1985) Intrauterine lead exposure and early development. In: Lekkas, T. D., ed. International conference: heavy metals in the environment; September; Athens, Greece, v. 2. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 153-155.

World Almanac and Book of Facts. (1987) United States population. New York, NY: Pharos/Scripps Howard; pp. 217-247.

World Health Organization. (1986) Regional Office for Europe: air quality guidelines. [Review draft, vol. II. Lead, ch. 19, pp.1-34].

Worth, D.; Matrange, A.; Lieberman, M.; DeVos, E.; Karelekas, P.; Ryan, C.; Craun, G. (1981) Lead in drinking water: the contribution of household tap water to blood lead levels. In: Lynam, D. R.; Piantanida, L. G.; Cole, J. F., eds. Proceedings of the 2nd international symposium on environmental lead research. New York, NY: Academic Press; pp. 199-225.

Yankel, A. J.; von Lindern, I. H.; Walter, S. D. (1977) The Silver Valley lead study: the relationship between childhood blood lead levels and environmental exposure. J. Air Pollut. Control Assoc. 27: 763-767.

Yip, R.; Norris, T. N.; Anderson, A. S. (1981) Iron status of children with elevated blood lead concentrations. J. Pediatr. 98: 922-925.

Yule, W.; Lansdown, R. (1983) Lead and children's development: recent findings. In: International conference: heavy metals in the environment: v. 2; September; Heidelberg, West Germany. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 912-916.

Yule, W.; Lansdown, R.; Millar, I. B.; Urbanowicz, M.-A. (1981) The relationship between blood lead concentrations, intelligence and attainment in a school population: a pilot study. Dev. Med. Child Neurol. 23: 567-576.

Yule, W.; Urbanowicz, M.-A.; Lansdown, R.; Millar, I. B. (1984) Teachers' ratings of children's behavior in relation to blood lead levels. Br. J. Dev. Psychol. 2: 295-305.

Zaric, M.; Prpic-Majic, D.; Kostial, K.; Piasek, M. (1987) Exposure to lead and reproduction. In: Summary proceedings of a workshop: selected aspects of exposure to heavy metals in the environment. Monitors, indicators, and high risk groups; April 1985. Washington, DC: National Academy of Sciences; Yugoslavia: Council of Academies of Sciences and Arts; pp. 119-126.

Ziegler, E. E.; Edwards, B. B.; Jensen, R. L.; Mahaffey, K. R.; Fomon, S. J. (1978) Absorption and retention of lead by infants. Pediatr. Res. 12: 29-34.

APPENDIX A

TABLES OF INDIVIDUAL SMSAs WITH A POPULATION OF OVER ONE MILLION SHOWING NUMBERS OF YOUNG CHILDREN BY THE AGE OF THEIR HOUSING AND FAMILY INCOME

This Appendix contains 38 tables for the SMSAs with populations of 1,000,000 or more. They appear in alphabetical order by the city that gives its name to the SMSA. One exception is Table A-21, for Nassau-Suffolk, NY showing only data for the total population in this SMSA, which had no city large enough to be defined as a central city.

The data come from tapes of 1980 U.S. Census enumerations, and cover children aged 6 months to 5 years of all races. Their distribution by residential status: "In Central City", "Not In Central City", and family income by age of residential unit is shown.

TABLE A- 1 ANAHEIM-SANTA ANA-GARDEN GROVE,

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	900	3200	300	4400	11.4	9.3	2.6	8.2
$6,000-$14,999	1300	10400	2900	14600	16.5	30.2	25.2	27.1
$15,000 OR MORE	5700	20800	8300	34800	72.2	60.5	72.2	64.7
TOTAL	7900	34400	11500	53800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	3000	2900	6400	8.9	5.8	6.0	6.0
$6,000-$14,999	1600	10800	5800	18200	28.6	21.0	11.9	17.2
$15,000 OR MORE	3500	37700	40000	81200	62.5	73.2	82.1	76.7
TOTAL	5600	51500	48700	105800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1400	6200	3200	10800	10.4	7.2	5.3	6.8
$6,000-$14,999	2900	21200	8700	32800	21.5	24.7	14.5	20.6
$15,000 OR MORE	9200	58500	48300	116000	68.1	68.1	80.2	72.7
TOTAL	13500	85900	60200	159600	100.0	100.0	100.0	100.0

TABLE A- 2 ATLANTA, GA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3000	7300	2900	13200	28.3	36.5	47.5	36.0
$6,000-$14,999	2700	6100	1400	10200	25.5	30.5	23.0	27.8
$15,000 OR MORE	4900	6600	1800	13300	46.2	33.0	29.5	36.2
TOTAL	10600	20000	6100	36700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1700	4900	3100	9700	15.0	9.4	4.1	6.9
$6,000-$14,999	3400	13600	12300	29300	30.1	26.2	16.1	21.0
$15,000 OR MORE	6200	33400	61100	100700	54.9	64.4	79.9	72.1
TOTAL	11300	51900	76500	139700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4700	12200	6000	22900	21.5	17.0	7.3	13.0
$6,000-$14,999	6100	19700	13700	39500	27.9	27.4	16.6	22.4
$15,000 OR MORE	11100	40000	62900	114000	50.7	55.6	76.2	64.6
TOTAL	21900	71900	82600	176400	100.0	100.0	100.0	100.0

TABLE A- 3 BALTIMORE, MD.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	7900	5400	1100	14400	21.1	26.9	30.6	23.6
$6,000-$14,999	13200	4900	1800	19900	35.3	24.4	50.0	32.6
$15,000 OR MORE	16300	9800	700	26800	43.6	48.8	19.4	43.9
TOTAL	37400	20100	3600	61100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1800	2500	3200	7500	9.3	6.6	6.8	7.2
$6,000-$14,999	6900	8100	5300	20300	35.8	21.4	11.2	19.4
$15,000 OR MORE	10600	27300	38900	76800	54.9	72.0	82.1	73.4
TOTAL	19300	37900	47400	104600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	9700	7900	4300	21900	17.1	13.6	8.4	13.2
$6,000-$14,999	20100	13000	7100	40200	35.4	22.4	13.9	24.3
$15,000 OR MORE	26900	37100	39600	103600	47.4	64.0	77.6	62.5
TOTAL	56700	58000	51000	165700	100.0	100.0	100.0	100.0

TABLE A- 4 BOSTON, MASS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	8500	2500	300	11300	31.7	36.8	13.6	31.6
$6,000-$14,999	8100	2500	1700	12300	30.2	36.8	77.3	34.4
$15,000 OR MORE	10200	1800	200	12200	38.1	26.5	9.1	34.1
TOTAL	26800	6800	2200	35800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	6100	2100	1100	9300	7.3	6.2	4.9	6.6
$6,000-$14,999	19700	5100	2500	27300	23.6	15.0	11.1	19.5
$15,000 OR MORE	57800	26900	19000	103700	69.1	78.9	84.1	73.9
TOTAL	83600	34100	22600	140300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	14600	4600	1400	20600	13.2	11.2	5.6	11.7
$6,000-$14,999	27800	7600	4200	39600	25.2	18.6	16.9	22.5
$15,000 OR MORE	68000	28700	19200	115900	61.6	70.2	77.4	65.8
TOTAL	110400	40900	24800	176100	100.0	100.0	100.0	100.0

TABLE A- 5 BUFFALO, N.Y.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	6200	500	0	6700	28.3	13.9	0.0	26.0
$6,000-$14,999	5500	2300	0	7800	25.1	63.9	0.0	30.2
$15,000 OR MORE	10200	800	300	11300	46.6	22.2	100.0	43.8
TOTAL	21900	3600	300	25800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	2600	1300	800	4700	10.4	5.3	5.0	7.2
$6,000-$14,999	6300	4000	1600	11900	25.3	16.3	10.0	18.2
$15,000 OR MORE	16000	19300	13600	48900	64.3	78.5	85.0	74.7
TOTAL	24900	24600	16000	65500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	8800	1800	800	11400	18.8	6.4	4.9	12.5
$6,000-$14,999	11800	6300	1600	19700	25.2	22.3	9.8	21.6
$15,000 OR MORE	26200	20100	13900	60200	56.0	71.3	85.3	65.9
TOTAL	46800	28200	16300	91300	100.0	100.0	100.0	100.0

TABLE A- 6 CHICAGO, ILL.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	41500	21700	4000	67200	21.7	29.1	31.7	24.1
$6,000-$14,999	53000	17000	2500	72500	27.7	22.8	19.8	26.1
$15,000 OR MORE	96600	35900	6100	138600	50.5	48.1	48.4	49.8
TOTAL	191100	74600	12600	278300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	5000	7200	5300	17500	6.2	5.0	4.2	5.0
$6,000-$14,999	13700	16200	10700	40600	17.0	11.3	8.5	11.6
$15,000 OR MORE	61700	120500	110200	292400	76.7	83.7	87.3	83.4
TOTAL	80400	143900	126200	350500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	46500	28900	9300	84700	17.1	13.2	6.7	13.5
$6,000-$14,999	66700	33200	13200	113100	24.6	15.2	9.5	18.0
$15,000 OR MORE	158300	156400	116300	431000	58.3	71.6	83.8	68.5
TOTAL	271500	218500	138800	628800	100.0	100.0	100.0	100.0

TABLE A- 7 CINCINNATI, OHIO-KY.-IND.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4200	4300	800	9300	21.8	35.2	38.1	27.7
$6,000-$14,999	4800	3400	300	8500	24.9	27.9	14.3	25.3
$15,000 OR MORE	10300	4500	1000	15800	53.4	36.9	47.6	47.0
TOTAL	19300	12200	2100	33600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	2200	2900	1900	7000	8.8	8.7	5.7	7.7
$6,000-$14,999	8000	4900	4700	17600	32.0	14.8	14.2	19.3
$15,000 OR MORE	14800	25400	26600	66800	59.2	76.5	80.1	73.1
TOTAL	25000	33200	33200	91400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	6400	7200	2700	16300	14.4	15.9	7.6	13.0
$6,000-$14,999	12800	8300	5000	26100	28.9	18.3	14.2	20.9
$15,000 OR MORE	25100	29900	27600	82600	56.7	65.9	78.2	66.1
TOTAL	44300	45400	35300	125000	100.0	100.0	100.0	100.0

TABLE A- 8 CLEVELAND, OHIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	10700	3200	500	14400	27.0	26.0	50.0	27.2
$6,000-$14,999	12400	3500	100	16000	31.3	28.5	10.0	30.2
$15,000 OR MORE	16500	5600	400	22500	41.7	45.5	40.0	42.5
TOTAL	39600	12300	1000	52900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	3100	2600	500	6200	8.7	6.3	2.2	6.2
$6,000-$14,999	5700	6500	2600	14800	16.1	15.7	11.2	14.8
$15,000 OR MORE	26700	32300	20100	79100	75.2	78.0	86.6	79.0
TOTAL	35500	41400	23200	100100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	13800	5800	1000	20600	18.4	10.8	4.1	13.5
$6,000-$14,999	18100	10000	2700	30800	24.1	18.6	11.2	20.1
$15,000 OR MORE	43200	37900	20500	101600	57.5	70.6	84.7	66.4
TOTAL	75100	53700	24200	153000	100.0	100.0	100.0	100.0

TABLE A- 9 COLUMBUS, OHIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3900	3800	1700	9400	22.7	17.7	10.8	17.3
$6,000-$14,999	6000	5700	4600	16300	34.9	26.5	29.3	30.0
$15,000 OR MORE	7300	12000	9400	28700	42.4	55.8	59.9	52.8
TOTAL	17200	21500	15700	54400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1200	400	400	2000	8.4	2.4	2.2	4.1
$6,000-$14,999	4200	3000	4500	11700	29.4	18.3	24.3	23.8
$15,000 OR MORE	8900	13000	13600	35500	62.2	79.3	73.5	72.2
TOTAL	14300	16400	18500	49200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5100	4200	2100	11400	16.2	11.1	6.1	11.0
$6,000-$14,999	10200	8700	9100	28000	32.4	23.0	26.6	27.0
$15,000 OR MORE	16200	25000	23000	64200	51.4	66.0	67.3	62.0
TOTAL	31500	37900	34200	103600	100.0	100.0	100.0	100.0

TABLE A- 10 DALLAS-FORT WORTH, TEX.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4100	9900	1800	15800	15.4	16.2	7.4	14.1
$6,000-$14,999	10700	18200	6000	34900	40.1	29.7	24.8	31.1
$15,000 OR MORE	11900	33200	16400	61500	44.6	54.2	67.8	54.8
TOTAL	26700	61300	24200	112200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	900	4600	3700	9200	6.3	7.9	4.5	5.9
$6,000-$14,999	4300	14600	11900	30800	30.3	25.0	14.4	19.8
$15,000 OR MORE	9000	39100	67100	115200	63.4	67.1	81.1	74.2
TOTAL	14200	58300	82700	155200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5000	14500	5500	25000	12.2	12.1	5.1	9.3
$6,000-$14,999	15000	32800	17900	65700	36.7	27.4	16.7	24.6
$15,000 OR MORE	20900	72300	83500	176700	51.1	60.5	78.1	66.1
TOTAL	40900	119600	106900	267400	100.0	100.0	100.0	100.0

TABLE A- 11 DENVER-BOULDER, COLO.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2900	2600	1200	6700	18.6	14.6	15.4	16.3
$6,000-$14,999	4200	5100	1500	10800	26.9	28.7	19.2	26.2
$15,000 OR MORE	8500	10100	5100	23700	54.5	56.7	65.4	57.5
TOTAL	15600	17800	7800	41200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1000	1400	3200	5600	10.3	4.4	5.3	5.5
$6,000-$14,999	3400	8100	7900	19400	35.1	25.4	13.1	19.1
$15,000 OR MORE	5300	22400	49000	76700	54.6	70.2	81.5	75.4
TOTAL	9700	31900	60100	101700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3900	4000	4400	12300	15.4	8.0	6.5	8.6
$6,000-$14,999	7600	13200	9400	30200	30.0	26.6	13.8	21.1
$15,000 OR MORE	13800	32500	54100	100400	54.5	65.4	79.7	70.3
TOTAL	25300	49700	67900	142900	100.0	100.0	100.0	100.0

TABLE A- 12 DETROIT, MICH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	16500	5700	800	23000	21.0	17.3	28.6	20.1
$6,000-$14,999	21200	9200	900	31300	27.0	28.0	32.1	27.4
$15,000 OR MORE	40900	18000	1100	60000	52.0	54.7	39.3	52.5
TOTAL	78600	32900	2800	114300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	5500	5600	3200	14300	8.7	5.0	3.7	5.5
$6,000-$14,999	12400	16400	9200	38000	19.6	14.8	10.6	14.5
$15,000 OR MORE	45400	89100	74700	209200	71.7	80.2	85.8	80.0
TOTAL	63300	111100	87100	261500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	22000	11300	4000	37300	15.5	7.8	4.4	9.9
$6,000-$14,999	33600	25600	10100	69300	23.7	17.8	11.2	18.4
$15,000 OR MORE	86300	107100	75800	269200	60.8	74.4	84.3	71.6
TOTAL	141900	144000	89900	375800	100.0	100.0	100.0	100.0

TABLE A- 13 FORT LAUDERDALE-HOLLYWOOD, FLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	600	200	800	0.0	6.4	4.3	5.0
$6,000-$14,999	1000	3400	1600	6000	47.6	36.2	34.8	37.3
$15,000 OR MORE	1100	5400	2800	9300	52.4	57.4	60.9	57.8
TOTAL	2100	9400	4600	16100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	2000	1500	3900	44.4	10.3	6.0	8.6
$6,000-$14,999	300	4500	4900	9700	33.3	23.2	19.7	21.5
$15,000 OR MORE	200	12900	18500	31600	22.2	66.5	74.3	69.9
TOTAL	900	19400	24900	45200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	400	2600	1700	4700	13.3	9.0	5.8	7.7
$6,000-$14,999	1300	7900	6500	15700	43.3	27.4	22.0	25.6
$15,000 OR MORE	1300	18300	21300	40900	43.3	63.5	72.2	66.7
TOTAL	3000	28800	29500	61300	100.0	100.0	100.0	100.0

TABLE A- 14 HOUSTON, TEX.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4800	11500	4700	21000	18.0	15.2	9.2	13.7
$6,000-$14,999	8200	18400	10300	36900	30.8	24.3	20.2	24.1
$15,000 OR MORE	13600	45900	35900	95400	51.1	60.6	70.5	62.2
TOTAL	26600	75800	50900	153300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1100	2300	4600	8000	11.2	6.4	4.9	5.7
$6,000-$14,999	2200	9400	10200	21800	22.4	26.2	10.8	15.6
$15,000 OR MORE	6500	24200	79600	110300	66.3	67.4	84.3	78.7
TOTAL	9800	35900	94400	140100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5900	13800	9300	29000	16.2	12.4	6.4	9.9
$6,000-$14,999	10400	27800	20500	58700	28.6	24.9	14.1	20.0
$15,000 OR MORE	20100	70100	115500	205700	55.2	62.8	79.5	70.1
TOTAL	36400	111700	145300	293400	100.0	100.0	100.0	100.0

TABLE A- 15 INDIANAPOLIS, IND.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3700	2700	1700	8100	18.1	9.8	12.9	13.3
$6,000-$14,999	5700	6900	2600	15200	27.9	25.1	19.7	24.9
$15,000 OR MORE	11000	17900	8900	37800	53.9	65.1	67.4	61.9
TOTAL	20400	27500	13200	61100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	600	700	500	1800	8.2	4.9	2.8	4.6
$6,000-$14,999	2400	2900	2200	7500	32.9	20.1	12.5	19.1
$15,000 OR MORE	4300	10800	14900	30000	58.9	75.0	84.7	76.3
TOTAL	7300	14400	17600	39300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4300	3400	2200	9900	15.5	8.1	7.1	9.9
$6,000-$14,999	8100	9800	4800	22700	29.2	23.4	15.6	22.6
$15,000 OR MORE	15300	28700	23800	67800	55.2	68.5	77.3	67.5
TOTAL	27700	41900	30800	100400	100.0	100.0	100.0	100.0

TABLE A- 16 KANSAS CITY, MO.-KANS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2700	2500	500	5700	18.1	19.4	12.2	17.9
$6,000-$14,999	5100	3100	1100	9300	34.2	24.0	26.8	29.2
$15,000 OR MORE	7100	7300	2500	16900	47.7	56.6	61.0	53.0
TOTAL	14900	12900	4100	31900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	3100	3200	1400	7700	18.7	9.4	4.7	9.6
$6,000-$14,999	4300	7300	4700	16300	25.9	21.5	15.8	20.3
$15,000 OR MORE	9200	23500	23700	56400	55.4	69.1	79.5	70.1
TOTAL	16600	34000	29800	80400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5800	5700	1900	13400	18.4	12.2	5.6	11.9
$6,000-$14,999	9400	10400	5800	25600	29.8	22.2	17.1	22.8
$15,000 OR MORE	16300	30800	26200	73300	51.7	65.7	77.3	65.3
TOTAL	31500	46900	33900	112300	100.0	100.0	100.0	100.0

TABLE A- 17 LOS ANGELES-LONG BEACH, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	24700	21100	3400	49200	20.1	15.8	11.5	17.2
$6,000-$14,999	43300	38800	8800	90900	35.3	29.0	29.8	31.8
$15,000 OR MORE	54700	73800	17300	145800	44.6	55.2	58.6	51.0
TOTAL	122700	133700	29500	285900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	11700	24600	5200	41500	11.4	11.2	8.1	10.7
$6,000-$14,999	29900	57900	11900	99700	29.0	26.3	18.5	25.7
$15,000 OR MORE	61400	137500	47300	246200	59.6	62.5	73.4	63.6
TOTAL	103000	220000	64400	387400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	36400	45700	8600	90700	16.1	12.9	9.2	13.5
$6,000-$14,999	73200	96700	20700	190600	32.4	27.3	22.0	28.3
$15,000 OR MORE	116100	211300	64600	392000	51.4	59.7	68.8	58.2
TOTAL	225700	353700	93900	673300	100.0	100.0	100.0	100.0

TABLE A- 18 MIAMI, FLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
URBAN STATUS/ FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3000	1800	500	5300	45.5	15.9	13.5	24.5
$6,000-$14,999	1800	5800	1600	9200	27.3	51.3	43.2	42.6
$15,000 OR MORE	1800	3700	1600	7100	27.3	32.7	43.2	32.9
TOTAL	6600	11300	3700	21600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1900	7100	5000	14000	26.4	14.6	12.9	14.8
$6,000-$14,999	1800	13100	9200	24100	25.0	27.0	23.7	25.5
$15,000 OR MORE	3500	28300	24600	56400	48.6	58.4	63.4	59.7
TOTAL	7200	48500	38800	94500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4900	8900	5500	19300	35.5	14.9	12.9	16.6
$6,000-$14,999	3600	18900	10800	33300	26.1	31.6	25.4	28.7
$15,000 OR MORE	5300	32000	26200	63500	38.4	53.5	61.6	54.7
TOTAL	13800	59800	42500	116100	100.0	100.0	100.0	100.0

TABLE A- 19 MILWAUKEE, WIS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	6800	2700	800	10300	19.1	12.8	13.8	16.5
$6,000-$14,999	8500	5000	1200	14700	23.9	23.7	20.7	23.5
$15,000 OR MORE	20300	13400	3800	37500	57.0	63.5	65.5	60.0
TOTAL	35600	21100	5800	62500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	900	500	2200	4.6	4.8	2.2	3.7
$6,000-$14,999	1600	2300	2200	6100	9.1	12.2	9.5	10.2
$15,000 OR MORE	15100	15700	20500	51300	86.3	83.1	88.4	86.1
TOTAL	17500	18900	23200	59600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	7600	3600	1300	12500	14.3	9.0	4.5	10.2
$6,000-$14,999	10100	7300	3400	20800	19.0	18.2	11.7	17.0
$15,000 OR MORE	35400	29100	24300	88800	66.7	72.7	83.8	72.7
TOTAL	53100	40000	29000	122100	100.0	100.0	100.0	100.0

TABLE A- 20 MINNEAPOLIS-ST. PAUL, MINN.-WIS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3700	1500	700	5900	9.9	16.1	14.6	11.5
$6,000-$14,999	6700	2600	1400	10700	18.0	28.0	29.2	20.9
$15,000 OR MORE	26800	5200	2700	34700	72.0	55.9	56.3	67.6
TOTAL	37200	9300	4800	51300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1100	2600	2900	6600	4.8	5.3	4.6	4.9
$6,000-$14,999	4100	6400	7800	18300	18.0	12.9	12.4	13.5
$15,000 OR MORE	17600	40500	52300	110400	77.2	81.8	83.0	81.6
TOTAL	22800	49500	63000	135300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4800	4100	3600	12500	8.0	7.0	5.3	6.7
$6,000-$14,999	10800	9000	9200	29000	18.0	15.3	13.6	15.5
$15,000 OR MORE	44400	45700	55000	145100	74.0	77.7	81.1	77.8
TOTAL	60000	58800	67800	186600	100.0	100.0	100.0	100.0

TABLE A- 21 NASSAU-SUFFOLK, NY

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	2800	6000	1700	10500	5.5	6.1	3.9	5.5
$6,000-$14,999	8100	13000	5700	26800	15.9	13.3	13.0	13.9
$15,000 OR MORE	40200	78700	36300	155200	78.7	80.6	83.1	80.6
TOTAL	51100	97700	43700	192500	100.0	100.0	100.0	100.0

TABLE A- 22 NEW ORLEANS, LA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	8600	9000	2100	19700	37.4	36.3	23.6	34.7
$6,000-$14,999	8000	5700	2100	15800	34.8	23.0	23.6	27.9
$15,000 OR MORE	6400	10100	4700	21200	27.8	40.7	52.8	37.4
TOTAL	23000	24800	8900	56700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1600	3400	2900	7900	28.6	13.9	9.3	12.9
$6,000-$14,999	1800	5700	4600	12100	32.1	23.3	14.7	19.7
$15,000 OR MORE	2200	15400	23700	41300	39.3	62.9	76.0	67.4
TOTAL	5600	24500	31200	61300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	10200	12400	5000	27600	35.7	25.2	12.5	23.4
$6,000-$14,999	9800	11400	6700	27900	34.3	23.1	16.7	23.6
$15,000 OR MORE	8600	25500	28400	62500	30.1	51.7	70.8	53.0
TOTAL	28600	49300	40100	118000	100.0	100.0	100.0	100.0

TABLE A- 23 NEW YORK, N.Y.-N.J.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	104600	37300	10300	152200	28.8	24.5	18.4	26.7
$6,000-$14,999	112200	45700	17100	175000	30.9	30.0	30.5	30.6
$15,000 OR MORE	146100	69100	28600	243800	40.3	45.4	51.1	42.7
TOTAL	362900	152100	56000	571000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	5100	2300	1700	9100	8.5	4.7	7.1	6.9
$6,000-$14,999	10600	5700	2800	19100	17.7	11.7	11.8	14.4
$15,000 OR MORE	44200	40800	19300	104300	73.8	83.6	81.1	78.7
TOTAL	59900	48800	23800	132500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	109700	39600	12000	161300	25.9	19.7	15.0	22.9
$6,000-$14,999	122800	51400	19900	194100	29.0	25.6	24.9	27.6
$15,000 OR MORE	190300	109900	47900	348100	45.0	54.7	60.0	49.5
TOTAL	422800	200900	79800	703500	100.0	100.0	100.0	100.0

TABLE A- 24 NEWARK, N.J.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	8500	5000	700	14200	38.1	48.1	36.8	41.0
$6,000-$14,999	7100	3400	400	10900	31.8	32.7	21.1	31.5
$15,000 OR MORE	6700	2000	800	9500	30.0	19.2	42.1	27.5
TOTAL	22300	10400	1900	34600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	5200	3100	600	8900	9.0	7.4	3.6	7.6
$6,000-$14,999	11600	5900	2200	19700	20.0	14.1	13.0	16.9
$15,000 OR MORE	41200	32700	14100	88000	71.0	78.4	83.4	75.5
TOTAL	58000	41700	16900	116600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	13700	8100	1300	23100	17.1	15.5	6.9	15.3
$6,000-$14,999	18700	9300	2600	30600	23.3	17.9	13.8	20.2
$15,000 OR MORE	47900	34700	14900	97500	59.7	66.6	79.3	64.5
TOTAL	80300	52100	18800	151200	100.0	100.0	100.0	100.0

TABLE A- 25 PHILADELPHIA, PA.-N.J.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	20700	7800	1100	29600	22.6	23.6	16.2	22.5
$6,000-$14,999	31200	8200	1600	41000	34.0	24.8	23.5	31.2
$15,000 OR MORE	39800	17000	4100	60900	43.4	51.5	60.3	46.3
TOTAL	91700	33000	6800	131500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	7300	6600	3700	17600	9.0	7.7	5.0	7.3
$6,000-$14,999	19300	17800	10000	47100	23.9	20.7	13.6	19.6
$15,000 OR MORE	54200	61600	59800	175600	67.1	71.6	81.4	73.1
TOTAL	80800	86000	73500	240300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	28000	14400	4800	47200	16.2	12.1	6.0	12.7
$6,000-$14,999	50500	26000	11600	88100	29.3	21.8	14.4	23.7
$15,000 OR MORE	94000	78600	63900	236500	54.5	66.1	79.6	63.6
TOTAL	172500	119000	80300	371800	100.0	100.0	100.0	100.0

TABLE A- 26 PHOENIX, ARIZ.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2400	4900	1800	9100	27.0	17.0	6.1	13.6
$6,000-$14,999	2700	9200	5400	17300	30.3	31.9	18.4	25.8
$15,000 OR MORE	3800	14700	22200	40700	42.7	51.0	75.5	60.7
TOTAL	8900	28800	29400	67100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	2500	2600	5600	16.1	11.8	7.1	9.2
$6,000-$14,999	1400	7200	8500	17100	45.2	34.0	23.3	28.1
$15,000 OR MORE	1200	11500	25400	38100	38.7	54.2	69.6	62.7
TOTAL	3100	21200	36500	60800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2900	7400	4400	14700	24.2	14.8	6.7	11.5
$6,000-$14,999	4100	16400	13900	34400	34.2	32.8	21.1	26.9
$15,000 OR MORE	5000	26200	47600	78800	41.7	52.4	72.2	61.6
TOTAL	12000	50000	65900	127900	100.0	100.0	100.0	100.0

TABLE A- 27 PITTSBURGH, PA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3100	2100	200	5400	17.6	39.6	11.8	22.0
$6,000-$14,999	4700	500	900	6100	26.7	9.4	52.9	24.8
$15,000 OR MORE	9800	2700	600	13100	55.7	50.9	35.3	53.3
TOTAL	17600	5300	1700	24600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	7400	2400	1100	10900	13.8	6.3	3.2	8.7
$6,000-$14,999	11800	5400	5400	22600	22.0	14.1	15.9	18.0
$15,000 OR MORE	34500	30500	27400	92400	64.2	79.6	80.8	73.4
TOTAL	53700	38300	33900	125900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	10500	4500	1300	16300	14.7	10.3	3.7	10.8
$6,000-$14,999	16500	5900	6300	28700	23.1	13.5	17.7	19.1
$15,000 OR MORE	44300	33200	28000	105500	62.1	76.1	78.7	70.1
TOTAL	71300	43600	35600	150500	100.0	100.0	100.0	100.0

TABLE A- 28 PORTLAND, OREG.-WASH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3400	1000	900	5300	18.6	13.9	36.0	18.9
$6,000-$14,999	4300	2500	200	7000	23.5	34.7	8.0	25.0
$15,000 OR MORE	10600	3700	1400	15700	57.9	51.4	56.0	56.1
TOTAL	18300	7200	2500	28000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1400	1700	5200	8300	9.6	7.1	12.0	10.1
$6,000-$14,999	2700	5700	8100	16500	18.5	23.8	18.7	20.2
$15,000 OR MORE	10500	16500	30000	57000	71.9	69.0	69.3	69.7
TOTAL	14600	23900	43300	81800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4800	2700	6100	13600	14.6	8.7	13.3	12.4
$6,000-$14,999	7000	8200	8300	23500	21.3	26.4	18.1	21.4
$15,000 OR MORE	21100	20200	31400	72700	64.1	65.0	68.6	66.2
TOTAL	32900	31100	45800	109800	100.0	100.0	100.0	100.0

TABLE A- 29 RIVERSIDE-SAN BERNARDINO-ONTARIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1200	2700	1000	4900	14.6	14.4	7.1	12.0
$6,000-$14,999	3600	4200	1600	9400	43.9	22.5	11.3	22.9
$15,000 OR MORE	3400	11800	11500	26700	41.5	63.1	81.6	65.1
TOTAL	8200	18700	14100	41000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	3300	5600	6000	14900	18.6	11.2	12.5	12.9
$6,000-$14,999	5900	14300	9800	30000	33.3	28.6	20.5	26.0
$15,000 OR MORE	8500	30100	32100	70700	48.0	60.2	67.0	61.2
TOTAL	17700	50000	47900	115600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4500	8300	7000	19800	17.4	12.1	11.3	12.6
$6,000-$14,999	9500	18500	11400	39400	36.7	26.9	18.4	25.2
$15,000 OR MORE	11900	41900	43600	97400	45.9	61.0	70.3	62.2
TOTAL	25900	68700	62000	156600	100.0	100.0	100.0	100.0

TABLE A- 30 SACRAMENTO, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
URBAN STATUS/ FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1700	1600	1600	4900	25.4	18.6	25.4	22.7
$6,000-$14,999	2400	3800	1300	7500	35.8	44.2	20.6	34.7
$15,000 OR MORE	2600	3200	3400	9200	38.8	37.2	54.0	42.6
TOTAL	6700	8600	6300	21600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	3700	3200	7700	10.8	14.2	10.3	11.9
$6,000-$14,999	2700	8200	6000	16900	36.5	31.5	19.3	26.2
$15,000 OR MORE	3900	14100	21900	39900	52.7	54.2	70.4	61.9
TOTAL	7400	26000	31100	64500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2500	5300	4800	12600	17.7	15.3	12.8	14.6
$6,000-$14,999	5100	12000	7300	24400	36.2	34.7	19.5	28.3
$15,000 OR MORE	6500	17300	25300	49100	46.1	50.0	67.6	57.0
TOTAL	14100	34600	37400	86100	100.0	100.0	100.0	100.0

TABLE A- 31 ST. LOUIS, MO.-ILL.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	6300	1700	1500	9500	21.1	23.9	65.2	24.2
$6,000-$14,999	10100	2600	400	13100	33.9	36.6	17.4	33.4
$15,000 OR MORE	13400	2800	400	16600	45.0	39.4	17.4	42.3
TOTAL	29800	7100	2300	39200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	5400	5200	2900	13500	13.7	7.6	5.2	8.2
$6,000-$14,999	9800	10900	9500	30200	24.9	15.8	16.9	18.4
$15,000 OR MORE	24200	52700	43800	120700	61.4	76.6	77.9	73.4
TOTAL	39400	68800	56200	164400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	11700	6900	4400	23000	16.9	9.1	7.5	11.3
$6,000-$14,999	19900	13500	9900	43300	28.8	17.8	16.9	21.3
$15,000 OR MORE	37600	55500	44200	137300	54.3	73.1	75.6	67.4
TOTAL	69200	75900	58500	203600	100.0	100.0	100.0	100.0

TABLE A- 32 SAN ANTONIO, TEX.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	6200	7600	4800	18600	32.3	19.3	17.8	21.8
$6,000-$14,999	8300	16100	8500	32900	43.2	41.0	31.6	38.5
$15,000 OR MORE	4700	15600	13600	33900	24.5	39.7	50.6	39.7
TOTAL	19200	39300	26900	85400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	1300	1400	3500	21.1	20.3	9.0	13.6
$6,000-$14,999	1900	2200	3100	7200	50.0	34.4	19.9	27.9
$15,000 OR MORE	1100	2900	11100	15100	28.9	45.3	71.2	58.5
TOTAL	3800	6400	15600	25800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	7000	8900	6200	22100	30.4	19.5	14.6	19.9
$6,000-$14,999	10200	18300	11600	40100	44.3	40.0	27.3	36.1
$15,000 OR MORE	5800	18500	24700	49000	25.2	40.5	58.1	44.1
TOTAL	23000	45700	42500	111200	100.0	100.0	100.0	100.0

TABLE A- 33 SAN DIEGO, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1600	3800	2100	7500	11.3	13.1	9.0	11.3
$6,000-$14,999	7000	8800	7500	23300	49.3	30.4	32.1	35.0
$15,000 OR MORE	5600	16300	13800	35700	39.4	56.4	59.0	53.7
TOTAL	14200	28900	23400	66500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1200	3400	3900	8500	12.4	9.5	9.1	9.6
$6,000-$14,999	3400	12600	11000	27000	35.1	35.2	25.7	30.6
$15,000 OR MORE	5100	19800	27900	52800	52.6	55.3	65.2	59.8
TOTAL	9700	35800	42800	88300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2800	7200	6000	16000	11.7	11.1	9.1	10.3
$6,000-$14,999	10400	21400	18500	50300	43.5	33.1	27.9	32.5
$15,000 OR MORE	10700	36100	41700	88500	44.8	55.8	63.0	57.2
TOTAL	23900	64700	66200	154800	100.0	100.0	100.0	100.0

TABLE A- 34 SAN FRANCISCO-OAKLAND, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	5500	4900	1400	11800	14.8	24.6	25.0	18.8
$6,000-$14,999	9200	6400	1000	16600	24.7	32.2	17.9	26.5
$15,000 OR MORE	22500	8600	3200	34300	60.5	43.2	57.1	54.7
TOTAL	37200	19900	5600	62700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	4800	7500	2200	14500	12.8	9.1	4.5	8.6
$6,000-$14,999	7400	17000	6800	31200	19.7	20.7	13.8	18.5
$15,000 OR MORE	25400	57500	40200	123100	67.6	70.1	81.7	72.9
TOTAL	37600	82000	49200	168800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	10300	12400	3600	26300	13.8	12.2	6.6	11.4
$6,000-$14,999	16600	23400	7800	47800	22.2	23.0	14.2	20.6
$15,000 OR MORE	47900	66100	43400	157400	64.0	64.9	79.2	68.0
TOTAL	74800	101900	54800	231500	100.0	100.0	100.0	100.0

TABLE A- 35 SAN JOSE, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	2400	900	3800	8.1	8.7	2.9	5.8
$6,000-$14,999	2800	5000	4000	11800	45.2	18.1	12.7	18.1
$15,000 OR MORE	2900	20300	26500	49700	46.8	73.3	84.4	76.1
TOTAL	6200	27700	31400	65300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	2000	500	2700	4.3	7.2	4.7	6.3
$6,000-$14,999	900	5400	1100	7400	19.6	19.4	10.3	17.1
$15,000 OR MORE	3500	20500	9100	33100	76.1	73.5	85.0	76.6
TOTAL	4600	27900	10700	43200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	700	4400	1400	6500	6.5	7.9	3.3	6.0
$6,000-$14,999	3700	10400	5100	19200	34.3	18.7	12.1	17.7
$15,000 OR MORE	6400	40800	35600	82800	59.3	73.4	84.6	76.3
TOTAL	10800	55600	42100	108500	100.0	100.0	100.0	100.0

TABLE A- 36 SEATTLE-EVERETT, WASH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1600	1300	100	3000	8.8	12.6	4.2	9.7
$6,000-$14,999	4800	2300	900	8000	26.4	22.3	37.5	25.9
$15,000 OR MORE	11800	6700	1400	19900	64.8	65.0	58.3	64.4
TOTAL	18200	10300	2400	30900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	900	2200	1900	5000	8.7	5.8	4.4	5.5
$6,000-$14,999	1700	5900	3600	11200	16.3	15.5	8.3	12.2
$15,000 OR MORE	7800	29900	37800	75500	75.0	78.7	87.3	82.3
TOTAL	10400	38000	43300	91700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2500	3500	2000	8000	8.7	7.2	4.4	6.5
$6,000-$14,999	6500	8200	4500	19200	22.7	17.0	9.8	15.7
$15,000 OR MORE	19600	36600	39200	95400	68.5	75.8	85.8	77.8
TOTAL	28600	48300	45700	122600	100.0	100.0	100.0	100.0

TABLE A- 37 TAMPA-ST. PETERSBURG, FLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2100	3400	1200	6700	24.1	19.0	19.0	20.4
$6,000-$14,999	4000	5900	1700	11600	46.0	33.0	27.0	35.3
$15,000 OR MORE	2600	8600	3400	14600	29.9	48.0	54.0	44.4
TOTAL	8700	17900	6300	32900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	3500	4200	8500	19.5	16.2	10.4	12.9
$6,000-$14,999	900	5900	11200	18000	22.0	27.3	27.9	27.3
$15,000 OR MORE	2400	12200	24800	39400	58.5	56.5	61.7	59.8
TOTAL	4100	21600	40200	65900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2900	6900	5400	15200	22.7	17.5	11.6	15.4
$6,000-$14,999	4900	11800	12900	29600	38.3	29.9	27.7	30.0
$15,000 OR MORE	5000	20800	28200	54000	39.1	52.7	60.6	54.7
TOTAL	12800	39500	46500	98800	100.0	100.0	100.0	100.0

TABLE A- 38 WASHINGTON, D.C.-MD.-VA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4300	4100	400	8800	20.4	26.6	9.3	21.6
$6,000-$14,999	5300	5000	1200	11500	25.1	32.5	27.9	28.2
$15,000 OR MORE	11500	6300	2700	20500	54.5	40.9	62.8	50.2
TOTAL	21100	15400	4300	40800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1800	5700	2300	9800	6.5	6.2	3.1	5.1
$6,000-$14,999	7900	16900	9400	34200	28.4	18.3	12.8	17.7
$15,000 OR MORE	18100	70000	61500	149600	65.1	75.6	84.0	77.3
TOTAL	27800	92600	73200	193600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	6100	9800	2700	18600	12.5	9.1	3.5	7.9
$6,000-$14,999	13200	21900	10600	45700	27.0	20.3	13.7	19.5
$15,000 OR MORE	29600	76300	64200	170100	60.5	70.6	82.8	72.6
TOTAL	48900	108000	77500	234400	100.0	100.0	100.0	100.0

APPENDIX B

TABLES OF INDIVIDUAL SMSAs WITH A POPULATION OF LESS THAN ONE MILLION SHOWING NUMBERS OF YOUNG CHILDREN BY THE AGE OF THEIR HOUSING AND FAMILY INCOME

The tables in this Appendix include 85 SMSAs with total populations of less than 1 million where the U.S. Census data permitted the population to be separated into two types of urban status, "In Central City" and "Not In Central City." They appear in alphabetical order by the city that gives its name to the SMSA.

The data come from tapes of 1980 U.S. Census enumerations, and cover children aged 6 months to 5 years of all races. Their distribution by residential status: "In Central City", "Not In Central City", and family income by age of residential unit is shown.

TABLE B- 1 AKRON, OHIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3400	1800	500	5700	24.5	26.5	26.3	25.2
$6,000-$14,999	2600	2000	600	5200	18.7	29.4	31.6	23.0
$15,000 OR MORE	7900	3000	800	11700	56.8	44.1	42.1	51.8
TOTAL	13900	6800	1900	22600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1000	700	1100	2800	11.1	5.0	9.7	8.2
$6,000-$14,999	2200	2200	2000	6400	24.4	15.7	17.7	18.7
$15,000 OR MORE	5800	11100	8200	25100	64.4	79.3	72.6	73.2
TOTAL	9000	14000	11300	34300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4400	2500	1600	8500	19.2	12.0	12.1	14.9
$6,000-$14,999	4800	4200	2600	11600	21.0	20.2	19.7	20.4
$15,000 OR MORE	13700	14100	9000	36800	59.8	67.8	68.2	64.7
TOTAL	22900	20800	13200	56900	100.0	100.0	100.0	100.0

TABLE B- 2 ALBANY-SCHENECTADY-TROY, N.Y.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3100	400	0	3500	23.1	14.3	0.0	20.3
$6,000-$14,999	3100	1000	400	4500	23.1	35.7	40.0	26.2
$15,000 OR MORE	7200	1400	600	9200	53.7	50.0	60.0	53.5
TOTAL	13400	2800	1000	17200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1400	900	200	2500	6.8	7.7	1.5	5.5
$6,000-$14,999	5900	2500	3400	11800	28.6	21.4	26.0	26.0
$15,000 OR MORE	13300	8300	9500	31100	64.6	70.9	72.5	68.5
TOTAL	20600	11700	13100	45400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4500	1300	200	6000	13.2	9.0	1.4	9.6
$6,000-$14,999	9000	3500	3800	16300	26.5	24.1	27.0	26.0
$15,000 OR MORE	20500	9700	10100	40300	60.3	66.9	71.6	64.4
TOTAL	34000	14500	14100	62600	100.0	100.0	100.0	100.0

TABLE B- 3 ALBUQUERQUE, N. MEX.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	800	2200	1400	4400	29.6	20.2	9.9	15.9
$6,000-$14,999	1000	3000	3700	7700	37.0	27.5	26.2	27.8
$15,000 OR MORE	900	5700	9000	15600	33.3	52.3	63.8	56.3
TOTAL	2700	10900	14100	27700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	900	700	1800	13.3	18.8	11.7	14.6
$6,000-$14,999	800	2100	2800	5700	53.3	43.8	46.7	46.3
$15,000 OR MORE	500	1800	2500	4800	33.3	37.5	41.7	39.0
TOTAL	1500	4800	6000	12300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	3100	2100	6200	23.8	19.7	10.4	15.5
$6,000-$14,999	1800	5100	6500	13400	42.9	32.5	32.3	33.5
$15,000 OR MORE	1400	7500	11500	20400	33.3	47.8	57.2	51.0
TOTAL	4200	15700	20100	40000	100.0	100.0	100.0	100.0

TABLE B- 4 ALLENTOWN-BETHLEHEM-EASTON, PA.-

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1700	500	100	2300	17.7	20.8	6.3	16.9
$6,000-$14,999	2600	0	200	2800	27.1	0.0	12.5	20.6
$15,000 OR MORE	5300	1900	1300	8500	55.2	79.2	81.3	62.5
TOTAL	9600	2400	1600	13600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	500	100	1100	4.2	7.4	1.0	3.8
$6,000-$14,999	3700	900	1100	5700	30.8	13.2	10.7	19.6
$15,000 OR MORE	7800	5400	9100	22300	65.0	79.4	88.3	76.6
TOTAL	12000	6800	10300	29100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2200	1000	200	3400	10.2	10.9	1.7	8.0
$6,000-$14,999	6300	900	1300	8500	29.2	9.8	10.9	19.9
$15,000 OR MORE	13100	7300	10400	30800	60.6	79.3	87.4	72.1
TOTAL	21600	9200	11900	42700	100.0	100.0	100.0	100.0

TABLE B- 5 ANN ARBOR, MICH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	300	400	700	0.0	5.9	20.0	7.5
$6,000-$14,999	300	900	100	1300	13.6	17.6	5.0	14.0
$15,000 OR MORE	1900	3900	1500	7300	86.4	76.5	75.0	78.5
TOTAL	2200	5100	2000	9300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	800	200	1200	7.1	12.1	4.8	8.8
$6,000-$14,999	700	500	800	2000	25.0	7.6	19.0	14.7
$15,000 OR MORE	1900	5300	3200	10400	67.9	80.3	76.2	76.5
TOTAL	2800	6600	4200	13600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	200	1100	600	1900	4.0	9.4	9.7	8.3
$6,000-$14,999	1000	1400	900	3300	20.0	12.0	14.5	14.4
$15,000 OR MORE	3800	9200	4700	17700	76.0	78.6	75.8	77.3
TOTAL	5000	11700	6200	22900	100.0	100.0	100.0	100.0

TABLE B- 6 APPLETON-OSHKOSH, WIS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	300	100	0	400	8.1	3.3	0.0	3.9
$6,000-$14,999	600	900	200	1700	16.2	30.0	5.6	16.5
$15,000 OR MORE	2800	2000	3400	8200	75.7	66.7	94.4	79.6
TOTAL	3700	3000	3600	10300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	700	300	200	1200	15.9	6.4	2.5	7.0
$6,000-$14,999	200	1000	1500	2700	4.5	21.3	18.5	15.7
$15,000 OR MORE	3500	3400	6400	13300	79.5	72.3	79.0	77.3
TOTAL	4400	4700	8100	17200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	400	200	1600	12.3	5.2	1.7	5.8
$6,000-$14,999	800	1900	1700	4400	9.9	24.7	14.5	16.0
$15,000 OR MORE	6300	5400	9800	21500	77.8	70.1	83.8	78.2
TOTAL	8100	7700	11700	27500	100.0	100.0	100.0	100.0

TABLE B- 7 AUSTIN, TEX.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	1300	600	2400	12.2	13.0	4.2	8.5
$6,000-$14,999	1700	3500	2800	8000	41.5	35.0	19.7	28.3
$15,000 OR MORE	1900	5200	10800	17900	46.3	52.0	76.1	63.3
TOTAL	4100	10000	14200	28300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	100	200	800	23.8	2.7	1.8	4.7
$6,000-$14,999	700	1600	3000	5300	33.3	43.2	26.3	30.8
$15,000 OR MORE	900	2000	8200	11100	42.9	54.1	71.9	64.5
TOTAL	2100	3700	11400	17200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	1400	800	3200	16.1	10.2	3.1	7.0
$6,000-$14,999	2400	5100	5800	13300	38.7	37.2	22.7	29.2
$15,000 OR MORE	2800	7200	19000	29000	45.2	52.6	74.2	63.7
TOTAL	6200	13700	25600	45500	100.0	100.0	100.0	100.0

TABLE B- 8 BAKERSFIELD, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	900	400	1300	0.0	17.3	6.9	10.0
$6,000-$14,999	1000	1500	900	3400	50.0	28.8	15.5	26.2
$15,000 OR MORE	1000	2800	4500	8300	50.0	53.8	77.6	63.8
TOTAL	2000	5200	5800	13000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1000	1800	1800	4600	13.0	10.3	18.0	13.1
$6,000-$14,999	3200	6100	2400	11700	41.6	34.9	24.0	33.2
$15,000 OR MORE	3500	9600	5800	18900	45.5	54.9	58.0	53.7
TOTAL	7700	17500	10000	35200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	2700	2200	5900	10.3	11.9	13.9	12.2
$6,000-$14,999	4200	7600	3300	15100	43.3	33.5	20.9	31.3
$15,000 OR MORE	4500	12400	10300	27200	46.4	54.6	65.2	56.4
TOTAL	9700	22700	15800	48200	100.0	100.0	100.0	100.0

TABLE B- 9 BATON ROUGE, LA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	2700	800	4100	21.4	25.7	15.1	22.0
$6,000-$14,999	1000	1800	1700	4500	35.7	17.1	32.1	24.2
$15,000 OR MORE	1200	6000	2800	10000	42.9	57.1	52.8	53.8
TOTAL	2800	10500	5300	18600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	1700	1400	3500	16.0	19.5	7.3	11.5
$6,000-$14,999	900	1700	2200	4800	36.0	19.5	11.4	15.7
$15,000 OR MORE	1200	5300	15700	22200	48.0	60.9	81.3	72.8
TOTAL	2500	8700	19300	30500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	4400	2200	7600	18.9	22.9	8.9	15.5
$6,000-$14,999	1900	3500	3900	9300	35.8	18.2	15.9	18.9
$15,000 OR MORE	2400	11300	18500	32200	45.3	58.9	75.2	65.6
TOTAL	5300	19200	24600	49100	100.0	100.0	100.0	100.0

TABLE B- 10 BEAUMONT-PORT ARTHUR-ORANGE, TEX

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2200	1100	400	3700	31.0	11.2	11.1	18.0
$6,000-$14,999	1800	2100	300	4200	25.4	21.4	8.3	20.5
$15,000 OR MORE	3100	6600	2900	12600	43.7	67.3	80.6	61.5
TOTAL	7100	9800	3600	20500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	200	100	700	19.0	2.7	1.6	4.4
$6,000-$14,999	500	1300	1000	2800	23.8	17.8	15.6	17.7
$15,000 OR MORE	1200	5800	5300	12300	57.1	79.5	82.8	77.8
TOTAL	2100	7300	6400	15800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2600	1300	500	4400	28.3	7.6	5.0	12.1
$6,000-$14,999	2300	3400	1300	7000	25.0	19.9	13.0	19.3
$15,000 OR MORE	4300	12400	8200	24900	46.7	72.5	82.0	68.6
TOTAL	9200	17100	10000	36300	100.0	100.0	100.0	100.0

TABLE B- 11 BIRMINGHAM, ALA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1400	2700	600	4700	15.9	21.1	17.1	18.7
$6,000-$14,999	3800	3700	1100	8600	43.2	28.9	31.4	34.3
$15,000 OR MORE	3600	6400	1800	11800	40.9	50.0	51.4	47.0
TOTAL	8800	12800	3500	25100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1700	2300	2300	6300	17.3	14.6	10.3	13.2
$6,000-$14,999	3600	4300	3900	11800	36.7	27.4	17.5	24.7
$15,000 OR MORE	4500	9100	16100	29700	45.9	58.0	72.2	62.1
TOTAL	9800	15700	22300	47800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3100	5000	2900	11000	16.7	17.5	11.2	15.1
$6,000-$14,999	7400	8000	5000	20400	39.8	28.1	19.4	28.0
$15,000 OR MORE	8100	15500	17900	41500	43.5	54.4	69.4	56.9
TOTAL	18600	28500	25800	72900	100.0	100.0	100.0	100.0

TABLE B- 12 BRIDGEPORT, CONN.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	2900	700	0	3600	33.7	22.6	0.0	30.5
$6,000-$14,999	2300	600	0	2900	26.7	19.4	0.0	24.6
$15,000 OR MORE	3400	1800	100	5300	39.5	58.1	100.0	44.9
TOTAL	8600	3100	100	11800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	200	300	1000	8.1	3.4	7.3	6.2
$6,000-$14,999	1600	500	500	2600	25.8	8.6	12.2	16.1
$15,000 OR MORE	4100	5100	3300	12500	66.1	87.9	80.5	77.6
TOTAL	6200	5800	4100	16100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3400	900	300	4600	23.0	10.1	7.1	16.5
$6,000-$14,999	3900	1100	500	5500	26.4	12.4	11.9	19.7
$15,000 OR MORE	7500	6900	3400	17800	50.7	77.5	81.0	63.8
TOTAL	14800	8900	4200	27900	100.0	100.0	100.0	100.0

TABLE B- 13 CHARLESTON-NORTH CHARLESTON, S.C

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	200	0	700	14.7	5.0	0.0	6.4
$6,000-$14,999	800	2400	1600	4800	23.5	60.0	45.7	44.0
$15,000 OR MORE	2100	1400	1900	5400	61.8	35.0	54.3	49.5
TOTAL	3400	4000	3500	10900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	800	1700	2900	13.3	9.4	9.3	9.8
$6,000-$14,999	1500	3700	5500	10700	50.0	43.5	30.2	36.0
$15,000 OR MORE	1100	4000	11000	16100	36.7	47.1	60.4	54.2
TOTAL	3000	8500	18200	29700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	900	1000	1700	3600	14.1	8.0	7.8	8.9
$6,000-$14,999	2300	6100	7100	15500	35.9	48.8	32.7	38.2
$15,000 OR MORE	3200	5400	12900	21500	50.0	43.2	59.4	53.0
TOTAL	6400	12500	21700	40600	100.0	100.0	100.0	100.0

TABLE B- 14 CHARLOTTE-GASTONIA, N.C.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	900	1500	0	2400	18.8	9.4	0.0	7.9
$6,000-$14,999	1000	5600	1800	8400	20.8	35.0	18.8	27.6
$15,000 OR MORE	2900	8900	7800	19600	60.4	55.6	81.3	64.5
TOTAL	4800	16000	9600	30400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	200	800	1400	9.5	2.4	7.0	5.8
$6,000-$14,999	1500	2400	2600	6500	35.7	28.9	22.6	27.1
$15,000 OR MORE	2300	5700	8100	16100	54.8	68.7	70.4	67.1
TOTAL	4200	8300	11500	24000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1300	1700	800	3800	14.4	7.0	3.8	7.0
$6,000-$14,999	2500	8000	4400	14900	27.8	32.9	20.9	27.4
$15,000 OR MORE	5200	14600	15900	35700	57.8	60.1	75.4	65.6
TOTAL	9000	24300	21100	54400	100.0	100.0	100.0	100.0

TABLE B- 15 CHATTANOOGA, TENN.-GA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1300	1800	300	3400	30.2	25.0	13.6	24.8
$6,000-$14,999	1800	1700	500	4000	41.9	23.6	22.7	29.2
$15,000 OR MORE	1200	3700	1400	6300	27.9	51.4	63.6	46.0
TOTAL	4300	7200	2200	13700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	100	900	1000	2000	3.4	12.2	7.8	8.6
$6,000-$14,999	1400	2300	4200	7900	48.3	31.1	32.6	34.1
$15,000 OR MORE	1400	4200	7700	13300	48.3	56.8	59.7	57.3
TOTAL	2900	7400	12900	23200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1400	2700	1300	5400	19.4	18.5	8.6	14.6
$6,000-$14,999	3200	4000	4700	11900	44.4	27.4	31.1	32.2
$15,000 OR MORE	2600	7900	9100	19600	36.1	54.1	60.3	53.1
TOTAL	7200	14600	15100	36900	100.0	100.0	100.0	100.0

TABLE B- 16 COLORADO SPRINGS, COLO.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	100	800	700	1600	5.9	12.1	7.4	9.0
$6,000-$14,999	1000	2800	3100	6900	58.8	42.4	32.6	38.8
$15,000 OR MORE	600	3000	5700	9300	35.3	45.5	60.0	52.2
TOTAL	1700	6600	9500	17800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	400	900	1500	20.0	7.5	16.7	12.8
$6,000-$14,999	100	2800	1300	4200	10.0	52.8	24.1	35.9
$15,000 OR MORE	700	2100	3200	6000	70.0	39.6	59.3	51.3
TOTAL	1000	5300	5400	11700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	300	1200	1600	3100	11.1	10.1	10.7	10.5
$6,000-$14,999	1100	5600	4400	11100	40.7	47.1	29.5	37.6
$15,000 OR MORE	1300	5100	8900	15300	48.1	42.9	59.7	51.9
TOTAL	2700	11900	14900	29500	100.0	100.0	100.0	100.0

TABLE B- 17 COLUMBIA, S.C.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	800	0	1400	23.1	24.2	0.0	21.9
$6,000-$14,999	700	1600	300	2600	26.9	48.5	60.0	40.6
$15,000 OR MORE	1300	900	200	2400	50.0	27.3	40.0	37.5
TOTAL	2600	3300	500	6400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	600	1500	2400	23.1	7.0	8.7	8.9
$6,000-$14,999	0	3100	4600	7700	0.0	36.0	26.7	28.4
$15,000 OR MORE	1000	4900	11100	17000	76.9	57.0	64.5	62.7
TOTAL	1300	8600	17200	27100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	900	1400	1500	3800	23.1	11.8	8.5	11.3
$6,000-$14,999	700	4700	4900	10300	17.9	39.5	27.7	30.7
$15,000 OR MORE	2300	5800	11300	19400	59.0	48.7	63.8	57.9
TOTAL	3900	11900	17700	33500	100.0	100.0	100.0	100.0

TABLE B- 18 DAVENPORT-ROCK ISLAND-MOLINE, IO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1500	500	500	2500	14.2	7.2	11.9	11.5
$6,000-$14,999	2400	1600	1200	5200	22.6	23.2	28.6	24.0
$15,000 OR MORE	6700	4800	2500	14000	63.2	69.6	59.5	64.5
TOTAL	10600	6900	4200	21700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	600	300	500	1400	11.5	4.9	7.6	7.8
$6,000-$14,999	800	600	700	2100	15.4	9.8	10.6	11.7
$15,000 OR MORE	3800	5200	5400	14400	73.1	85.2	81.8	80.4
TOTAL	5200	6100	6600	17900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2100	800	1000	3900	13.3	6.2	9.3	9.8
$6,000-$14,999	3200	2200	1900	7300	20.3	16.9	17.6	18.4
$15,000 OR MORE	10500	10000	7900	28400	66.5	76.9	73.1	71.7
TOTAL	15800	13000	10800	39600	100.0	100.0	100.0	100.0

TABLE B- 19 DAYTON, OHIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2000	1400	1100	4500	17.4	26.4	61.1	24.2
$6,000-$14,999	4100	2100	500	6700	35.7	39.6	27.8	36.0
$15,000 OR MORE	5400	1800	200	7400	47.0	34.0	11.1	39.8
TOTAL	11500	5300	1800	18600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1800	1700	1000	4500	15.9	7.0	6.5	8.9
$6,000-$14,999	3100	4000	3400	10500	27.4	16.5	22.2	20.7
$15,000 OR MORE	6400	18500	10900	35800	56.6	76.4	71.2	70.5
TOTAL	11300	24200	15300	50800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3800	3100	2100	9000	16.7	10.5	12.3	13.0
$6,000-$14,999	7200	6100	3900	17200	31.6	20.7	22.8	24.8
$15,000 OR MORE	11800	20300	11100	43200	51.8	68.8	64.9	62.2
TOTAL	22800	29500	17100	69400	100.0	100.0	100.0	100.0

TABLE B- 20 DES MOINES, IOWA

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	900	800	300	2000	14.3	15.1	9.7	13.6
$6,000-$14,999	1300	900	500	2700	20.6	17.0	16.1	18.4
$15,000 OR MORE	4100	3600	2300	10000	65.1	67.9	74.2	68.0
TOTAL	6300	5300	3100	14700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	300	100	700	25.0	6.5	1.5	5.6
$6,000-$14,999	200	1300	400	1900	16.7	28.3	6.0	15.2
$15,000 OR MORE	700	3000	6200	9900	58.3	65.2	92.5	79.2
TOTAL	1200	4600	6700	12500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1200	1100	400	2700	16.0	11.1	4.1	9.9
$6,000-$14,999	1500	2200	900	4600	20.0	22.2	9.2	16.9
$15,000 OR MORE	4800	6600	8500	19900	64.0	66.7	86.7	73.2
TOTAL	7500	9900	9800	27200	100.0	100.0	100.0	100.0

TABLE B- 21 DULUTH-SUPERIOR, MINN.-WIS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1300	0	100	1400	15.5	0.0	10.0	11.7
$6,000-$14,999	2900	800	400	4100	34.5	30.8	40.0	34.2
$15,000 OR MORE	4200	1800	500	6500	50.0	69.2	50.0	54.2
TOTAL	8400	2600	1000	12000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	100	100	400	4.8	5.9	1.4	3.1
$6,000-$14,999	900	100	1100	2100	21.4	5.9	15.5	16.2
$15,000 OR MORE	3100	1500	5900	10500	73.8	88.2	83.1	80.8
TOTAL	4200	1700	7100	13000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1500	100	200	1800	11.9	2.3	2.5	7.2
$6,000-$14,999	3800	900	1500	6200	30.2	20.9	18.5	24.8
$15,000 OR MORE	7300	3300	6400	17000	57.9	76.7	79.0	68.0
TOTAL	12600	4300	8100	25000	100.0	100.0	100.0	100.0

TABLE B- 22 ERIE, PA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	700	900	0	1600	13.7	32.1	0.0	17.4
$6,000-$14,999	1600	200	500	2300	31.4	7.1	38.5	25.0
$15,000 OR MORE	2800	1700	800	5300	54.9	60.7	61.5	57.6
TOTAL	5100	2800	1300	9200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	400	300	1500	16.3	12.5	5.6	11.1
$6,000-$14,999	900	800	1100	2800	18.4	25.0	20.4	20.7
$15,000 OR MORE	3200	2000	4000	9200	65.3	62.5	74.1	68.1
TOTAL	4900	3200	5400	13500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1500	1300	300	3100	15.0	21.7	4.5	13.7
$6,000-$14,999	2500	1000	1600	5100	25.0	16.7	23.9	22.5
$15,000 OR MORE	6000	3700	4800	14500	60.0	61.7	71.6	63.9
TOTAL	10000	6000	6700	22700	100.0	100.0	100.0	100.0

TABLE B- 23 EUGENE-SPRINGFIELD, OREG.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	300	700	800	1800	27.3	28.0	13.1	18.6
$6,000-$14,999	500	200	1400	2100	45.5	8.0	23.0	21.6
$15,000 OR MORE	300	1600	3900	5800	27.3	64.0	63.9	59.8
TOTAL	1100	2500	6100	9700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	300	300	900	10.3	7.9	4.8	6.9
$6,000-$14,999	1000	1600	800	3400	34.5	42.1	12.7	26.2
$15,000 OR MORE	1600	1900	5200	8700	55.2	50.0	82.5	66.9
TOTAL	2900	3800	6300	13000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	600	1000	1100	2700	15.0	15.9	8.9	11.9
$6,000-$14,999	1500	1800	2200	5500	37.5	28.6	17.7	24.2
$15,000 OR MORE	1900	3500	9100	14500	47.5	55.6	73.4	63.9
TOTAL	4000	6300	12400	22700	100.0	100.0	100.0	100.0

TABLE B- 24 EVANSVILLE, IND.-KY.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	800	600	200	1600	12.7	20.0	14.3	15.0
$6,000-$14,999	2300	900	500	3700	36.5	30.0	35.7	34.6
$15,000 OR MORE	3200	1500	700	5400	50.8	50.0	50.0	50.5
TOTAL	6300	3000	1400	10700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	200	100	500	8.3	4.4	1.3	3.4
$6,000-$14,999	700	700	1400	2800	29.2	15.6	17.9	19.0
$15,000 OR MORE	1500	3600	6300	11400	62.5	80.0	80.8	77.6
TOTAL	2400	4500	7800	14700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	800	300	2100	11.5	10.7	3.3	8.3
$6,000-$14,999	3000	1600	1900	6500	34.5	21.3	20.7	25.6
$15,000 OR MORE	4700	5100	7000	16800	54.0	68.0	76.1	66.1
TOTAL	8700	7500	9200	25400	100.0	100.0	100.0	100.0

TABLE B- 25 FLINT, MICH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2000	1200	400	3600	21.3	15.2	50.0	19.9
$6,000-$14,999	2300	2300	200	4800	24.5	29.1	25.0	26.5
$15,000 OR MORE	5100	4400	200	9700	54.3	55.7	25.0	53.6
TOTAL	9400	7900	800	18100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1100	1400	200	2700	11.5	11.3	1.6	7.8
$6,000-$14,999	2200	2100	2200	6500	22.9	16.9	17.6	18.8
$15,000 OR MORE	6300	8900	10100	25300	65.6	71.8	80.8	73.3
TOTAL	9600	12400	12500	34500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3100	2600	600	6300	16.3	12.8	4.5	12.0
$6,000-$14,999	4500	4400	2400	11300	23.7	21.7	18.0	21.5
$15,000 OR MORE	11400	13300	10300	35000	60.0	65.5	77.4	66.5
TOTAL	19000	20300	13300	52600	100.0	100.0	100.0	100.0

TABLE B- 26 FORT WAYNE, IND.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2100	200	300	2600	22.8	3.4	20.0	15.8
$6,000-$14,999	2900	1800	200	4900	31.5	31.0	13.3	29.7
$15,000 OR MORE	4200	3800	1000	9000	45.7	65.5	66.7	54.5
TOTAL	9200	5800	1500	16500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	900	0	600	1500	12.3	0.0	7.1	7.1
$6,000-$14,999	1900	900	1200	4000	26.0	16.4	14.3	18.9
$15,000 OR MORE	4500	4600	6600	15700	61.6	83.6	78.6	74.1
TOTAL	7300	5500	8400	21200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3000	200	900	4100	18.2	1.8	9.1	10.9
$6,000-$14,999	4800	2700	1400	8900	29.1	23.9	14.1	23.6
$15,000 OR MORE	8700	8400	7600	24700	52.7	74.3	76.8	65.5
TOTAL	16500	11300	9900	37700	100.0	100.0	100.0	100.0

TABLE B- 27 FRESNO, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	2700	800	4000	9.8	28.4	11.0	18.3
$6,000-$14,999	1400	2500	2000	5900	27.5	26.3	27.4	26.9
$15,000 OR MORE	3200	4300	4500	12000	62.7	45.3	61.6	54.8
TOTAL	5100	9500	7300	21900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1400	1500	1000	3900	18.9	14.4	11.4	14.7
$6,000-$14,999	2700	4300	2500	9500	36.5	41.3	28.4	35.7
$15,000 OR MORE	3300	4600	5300	13200	44.6	44.2	60.2	49.6
TOTAL	7400	10400	8800	26600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1900	4200	1800	7900	15.2	21.1	11.2	16.3
$6,000-$14,999	4100	6800	4500	15400	32.8	34.2	28.0	31.8
$15,000 OR MORE	6500	8900	9800	25200	52.0	44.7	60.9	52.0
TOTAL	12500	19900	16100	48500	100.0	100.0	100.0	100.0

TABLE B- 28 GARY-HAMMOND-EAST CHICAGO, IND.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3100	3300	400	6800	17.8	25.0	15.4	20.5
$6,000-$14,999	3200	1500	900	5600	18.4	11.4	34.6	16.9
$15,000 OR MORE	11100	8400	1300	20800	63.8	63.6	50.0	62.7
TOTAL	17400	13200	2600	33200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1100	800	900	2800	15.7	6.4	7.0	8.7
$6,000-$14,999	300	1400	600	2300	4.3	11.2	4.7	7.1
$15,000 OR MORE	5600	10300	11300	27200	80.0	82.4	88.3	84.2
TOTAL	7000	12500	12800	32300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4200	4100	1300	9600	17.2	16.0	8.4	14.7
$6,000-$14,999	3500	2900	1500	7900	14.3	11.3	9.7	12.1
$15,000 OR MORE	16700	18700	12600	48000	68.4	72.8	81.8	73.3
TOTAL	24400	25700	15400	65500	100.0	100.0	100.0	100.0

TABLE B- 29 GRAND RAPIDS, MICH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1500	200	400	2100	12.6	5.1	23.5	12.0
$6,000-$14,999	4400	700	500	5600	37.0	17.9	29.4	32.0
$15,000 OR MORE	6000	3000	800	9800	50.4	76.9	47.1	56.0
TOTAL	11900	3900	1700	17500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	700	300	300	1300	7.7	2.1	1.9	3.3
$6,000-$14,999	2000	2000	2900	6900	22.0	14.3	17.9	17.6
$15,000 OR MORE	6400	11700	13000	31100	70.3	83.6	80.2	79.1
TOTAL	9100	14000	16200	39300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2200	500	700	3400	10.5	2.8	3.9	6.0
$6,000-$14,999	6400	2700	3400	12500	30.5	15.1	19.0	22.0
$15,000 OR MORE	12400	14700	13800	40900	59.0	82.1	77.1	72.0
TOTAL	21000	17900	17900	56800	100.0	100.0	100.0	100.0

TABLE B- 30 GREENVILLE-SPARTANBURG, S.C.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	600	700	1800	29.4	18.8	33.3	25.7
$6,000-$14,999	700	1300	700	2700	41.2	40.6	33.3	38.6
$15,000 OR MORE	500	1300	700	2500	29.4	40.6	33.3	35.7
TOTAL	1700	3200	2100	7000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	900	1500	2900	8.2	6.3	7.4	7.1
$6,000-$14,999	2600	4100	4400	11100	42.6	28.7	21.6	27.2
$15,000 OR MORE	3000	9300	14500	26800	49.2	65.0	71.1	65.7
TOTAL	6100	14300	20400	40800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	1500	2200	4700	12.8	8.6	9.8	9.8
$6,000-$14,999	3300	5400	5100	13800	42.3	30.9	22.7	28.9
$15,000 OR MORE	3500	10600	15200	29300	44.9	60.6	67.6	61.3
TOTAL	7800	17500	22500	47800	100.0	100.0	100.0	100.0

TABLE B- 31 HARTFORD, CONN.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3600	500	600	4700	36.4	20.8	60.0	35.3
$6,000-$14,999	3600	1300	100	5000	36.4	54.2	10.0	37.6
$15,000 OR MORE	2700	600	300	3600	27.3	25.0	30.0	27.1
TOTAL	9900	2400	1000	13300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	600	600	0	1200	5.0	3.7	0.0	3.1
$6,000-$14,999	3200	2200	1000	6400	26.7	13.7	9.1	16.4
$15,000 OR MORE	8200	13200	10000	31400	68.3	82.5	90.9	80.5
TOTAL	12000	16000	11000	39000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4200	1100	600	5900	19.2	6.0	5.0	11.3
$6,000-$14,999	6800	3500	1100	11400	31.1	19.0	9.2	21.8
$15,000 OR MORE	10900	13800	10300	35000	49.8	75.0	85.8	66.9
TOTAL	21900	18400	12000	52300	100.0	100.0	100.0	100.0

TABLE B- 32 HONOLULU, HAWAII

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1100	1400	700	3200	22.0	10.8	9.0	12.4
$6,000-$14,999	1200	4400	3700	9300	24.0	33.8	47.4	36.0
$15,000 OR MORE	2700	7200	3400	13300	54.0	55.4	43.6	51.6
TOTAL	5000	13000	7800	25800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	2500	1000	3700	4.8	11.3	5.8	8.5
$6,000-$14,999	1800	6900	4600	13300	42.9	31.1	26.7	30.5
$15,000 OR MORE	2200	12800	11600	26600	52.4	57.7	67.4	61.0
TOTAL	4200	22200	17200	43600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1300	3900	1700	6900	14.1	11.1	6.8	9.9
$6,000-$14,999	3000	11300	8300	22600	32.6	32.1	33.2	32.6
$15,000 OR MORE	4900	20000	15000	39900	53.3	56.8	60.0	57.5
TOTAL	9200	35200	25000	69400	100.0	100.0	100.0	100.0

TABLE B- 33 HUNTSVILLE, ALA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	1900	200	2100	0.0	23.5	9.1	19.3
$6,000-$14,999	300	2100	500	2900	50.0	25.9	22.7	26.6
$15,000 OR MORE	300	4100	1500	5900	50.0	50.6	68.2	54.1
TOTAL	600	8100	2200	10900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	300	500	1300	20.0	5.1	6.9	8.3
$6,000-$14,999	1300	2700	2300	6300	52.0	45.8	31.9	40.4
$15,000 OR MORE	700	2900	4400	8000	28.0	49.2	61.1	51.3
TOTAL	2500	5900	7200	15600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	500	2200	700	3400	16.1	15.7	7.4	12.8
$6,000-$14,999	1600	4800	2800	9200	51.6	34.3	29.8	34.7
$15,000 OR MORE	1000	7000	5900	13900	32.3	50.0	62.8	52.5
TOTAL	3100	14000	9400	26500	100.0	100.0	100.0	100.0

TABLE B- 34 JACKSON, MISS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	1200	500	2300	18.8	11.7	10.9	12.7
$6,000-$14,999	1400	3800	1500	6700	43.8	36.9	32.6	37.0
$15,000 OR MORE	1200	5300	2600	9100	37.5	51.5	56.5	50.3
TOTAL	3200	10300	4600	18100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	800	500	1300	0.0	22.9	6.8	10.3
$6,000-$14,999	1000	800	2000	3800	55.6	22.9	27.4	30.2
$15,000 OR MORE	800	1900	4800	7500	44.4	54.3	65.8	59.5
TOTAL	1800	3500	7300	12600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	600	2000	1000	3600	12.0	14.5	8.4	11.7
$6,000-$14,999	2400	4600	3500	10500	48.0	33.3	29.4	34.2
$15,000 OR MORE	2000	7200	7400	16600	40.0	52.2	62.2	54.1
TOTAL	5000	13800	11900	30700	100.0	100.0	100.0	100.0

TABLE B- 35 JACKSONVILLE, FLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3700	4000	3700	11400	42.0	15.6	19.2	21.2
$6,000-$14,999	2600	9000	6300	17900	29.5	35.2	32.6	33.3
$15,000 OR MORE	2500	12600	9300	24400	28.4	49.2	48.2	45.4
TOTAL	8800	25600	19300	53700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	500	1400	2200	13.6	9.1	14.7	12.8
$6,000-$14,999	1200	1700	3200	6100	54.5	30.9	33.7	35.5
$15,000 OR MORE	700	3300	4900	8900	31.8	60.0	51.6	51.7
TOTAL	2200	5500	9500	17200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4000	4500	5100	13600	36.4	14.5	17.7	19.2
$6,000-$14,999	3800	10700	9500	24000	34.5	34.4	33.0	33.9
$15,000 OR MORE	3200	15900	14200	33300	29.1	51.1	49.3	47.0
TOTAL	11000	31100	28800	70900	100.0	100.0	100.0	100.0

TABLE B- 36 JERSEY CITY, N.J.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	5100	1500	200	6800	29.1	33.3	15.4	29.2
$6,000-$14,999	4600	1600	100	6300	26.3	35.6	7.7	27.0
$15,000 OR MORE	7800	1400	1000	10200	44.6	31.1	76.9	43.8
TOTAL	17500	4500	1300	23300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	3400	1100	100	4600	20.4	27.5	3.2	19.3
$6,000-$14,999	4800	1000	800	6600	28.7	25.0	25.8	27.7
$15,000 OR MORE	8500	1900	2200	12600	50.9	47.5	71.0	52.9
TOTAL	16700	4000	3100	23800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	8500	2600	300	11400	24.9	30.6	6.8	24.2
$6,000-$14,999	9400	2600	900	12900	27.5	30.6	20.5	27.4
$15,000 OR MORE	16300	3300	3200	22800	47.7	38.8	72.7	48.4
TOTAL	34200	8500	4400	47100	100.0	100.0	100.0	100.0

TABLE B- 37 KALAMAZOO-PORTAGE, MICH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	400	800	300	1500	12.1	20.0	15.0	16.1
$6,000-$14,999	1100	900	0	2000	33.3	22.5	0.0	21.5
$15,000 OR MORE	1800	2300	1700	5800	54.5	57.5	85.0	62.4
TOTAL	3300	4000	2000	9300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	800	100	1200	5.9	20.5	1.6	7.8
$6,000-$14,999	1200	1700	1500	4400	23.5	43.6	23.4	28.6
$15,000 OR MORE	3600	1400	4800	9800	70.6	35.9	75.0	63.6
TOTAL	5100	3900	6400	15400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	700	1600	400	2700	8.3	20.3	4.8	10.9
$6,000-$14,999	2300	2600	1500	6400	27.4	32.9	17.9	25.9
$15,000 OR MORE	5400	3700	6500	15600	64.3	46.8	77.4	63.2
TOTAL	8400	7900	8400	24700	100.0	100.0	100.0	100.0

TABLE B- 38 KNOXVILLE, TENN.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1300	1100	1500	3900	29.5	26.2	46.9	33.1
$6,000-$14,999	1900	1700	700	4300	43.2	40.5	21.9	36.4
$15,000 OR MORE	1200	1400	1000	3600	27.3	33.3	31.3	30.5
TOTAL	4400	4200	3200	11800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	600	600	1100	2300	14.6	8.8	9.0	10.0
$6,000-$14,999	900	1800	1500	4200	22.0	26.5	12.3	18.2
$15,000 OR MORE	2600	4400	9600	16600	63.4	64.7	78.7	71.9
TOTAL	4100	6800	12200	23100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1900	1700	2600	6200	22.4	15.5	16.9	17.8
$6,000-$14,999	2800	3500	2200	8500	32.9	31.8	14.3	24.4
$15,000 OR MORE	3800	5800	10600	20200	44.7	52.7	68.8	57.9
TOTAL	8500	11000	15400	34900	100.0	100.0	100.0	100.0

TABLE B- 39 LANSING-EAST LANSING, MICH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	1000	700	2300	8.6	19.6	26.9	15.6
$6,000-$14,999	1800	1400	700	3900	25.7	27.5	26.9	26.5
$15,000 OR MORE	4600	2700	1200	8500	65.7	52.9	46.2	57.8
TOTAL	7000	5100	2600	14700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	100	1100	1600	3.8	1.4	8.9	5.4
$6,000-$14,999	2900	1900	1600	6400	27.6	27.5	12.9	21.5
$15,000 OR MORE	7200	4900	9700	21800	68.6	71.0	78.2	73.2
TOTAL	10500	6900	12400	29800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	1100	1800	3900	5.7	9.2	12.0	8.8
$6,000-$14,999	4700	3300	2300	10300	26.9	27.5	15.3	23.1
$15,000 OR MORE	11800	7600	10900	30300	67.4	63.3	72.7	68.1
TOTAL	17500	12000	15000	44500	100.0	100.0	100.0	100.0

TABLE B- 40 LAS VEGAS, NEV.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	700	600	1300	0.0	9.5	9.2	9.2
$6,000-$14,999	0	2100	1000	3100	0.0	28.4	15.4	22.0
$15,000 OR MORE	200	4600	4900	9700	100.0	62.2	75.4	68.8
TOTAL	200	7400	6500	14100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	500	1400	2200	20.0	6.3	8.5	8.5
$6,000-$14,999	700	2900	3000	6600	46.7	36.2	18.3	25.5
$15,000 OR MORE	500	4600	12000	17100	33.3	57.5	73.2	66.0
TOTAL	1500	8000	16400	25900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	300	1200	2000	3500	17.6	7.8	8.7	8.7
$6,000-$14,999	700	5000	4000	9700	41.2	32.5	17.5	24.2
$15,000 OR MORE	700	9200	16900	26800	41.2	59.7	73.8	67.0
TOTAL	1700	15400	22900	40000	100.0	100.0	100.0	100.0

TABLE B- 41 LAWRENCE-HAVERHILL, MASS.-N.H.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1700	200	100	2000	25.0	8.7	10.0	19.8
$6,000-$14,999	1600	900	100	2600	23.5	39.1	10.0	25.7
$15,000 OR MORE	3500	1200	800	5500	51.5	52.2	80.0	54.5
TOTAL	6800	2300	1000	10100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1000	200	0	1200	19.6	5.3	0.0	10.2
$6,000-$14,999	700	400	0	1100	13.7	10.5	0.0	9.3
$15,000 OR MORE	3400	3200	2900	9500	66.7	84.2	100.0	80.5
TOTAL	5100	3800	2900	11800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2700	400	100	3200	22.7	6.6	2.6	14.6
$6,000-$14,999	2300	1300	100	3700	19.3	21.3	2.6	16.9
$15,000 OR MORE	6900	4400	3700	15000	58.0	72.1	94.9	68.5
TOTAL	11900	6100	3900	21900	100.0	100.0	100.0	100.0

TABLE B- 42 LEXINGTON-FAYETTE, KY.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	1100	1000	2600	11.4	15.3	16.7	14.8
$6,000-$14,999	1500	1500	1000	4000	34.1	20.8	16.7	22.7
$15,000 OR MORE	2400	4600	4000	11000	54.5	63.9	66.7	62.5
TOTAL	4400	7200	6000	17600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1200	400	900	2500	22.6	17.4	19.1	20.3
$6,000-$14,999	1100	1100	1200	3400	20.8	47.8	25.5	27.6
$15,000 OR MORE	3000	800	2600	6400	56.6	34.8	55.3	52.0
TOTAL	5300	2300	4700	12300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1700	1500	1900	5100	17.5	15.8	17.8	17.1
$6,000-$14,999	2600	2600	2200	7400	26.8	27.4	20.6	24.7
$15,000 OR MORE	5400	5400	6600	17400	55.7	56.8	61.7	58.2
TOTAL	9700	9500	10700	29900	100.0	100.0	100.0	100.0

TABLE B- 43 LORAIN-ELYRIA, OHIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1200	700	800	2700	21.1	14.9	18.6	18.4
$6,000-$14,999	1100	1000	800	2900	19.3	21.3	18.6	19.7
$15,000 OR MORE	3400	3000	2700	9100	59.6	63.8	62.8	61.9
TOTAL	5700	4700	4300	14700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	0	0	400	18.2	0.0	0.0	4.0
$6,000-$14,999	400	100	300	800	18.2	3.2	6.5	8.1
$15,000 OR MORE	1400	3000	4300	8700	63.6	96.8	93.5	87.9
TOTAL	2200	3100	4600	9900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1600	700	800	3100	20.3	9.0	9.0	12.6
$6,000-$14,999	1500	1100	1100	3700	19.0	14.1	12.4	15.0
$15,000 OR MORE	4800	6000	7000	17800	60.8	76.9	78.7	72.4
TOTAL	7900	7800	8900	24600	100.0	100.0	100.0	100.0

TABLE B- 44 LOUISVILLE, KY.-IND.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4600	2100	600	7300	38.0	24.7	35.3	32.7
$6,000-$14,999	3100	2200	600	5900	25.6	25.9	35.3	26.5
$15,000 OR MORE	4400	4200	500	9100	36.4	49.4	29.4	40.8
TOTAL	12100	8500	1700	22300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	2300	2000	5100	10.4	9.4	7.8	8.8
$6,000-$14,999	2000	4800	5000	11800	26.0	19.7	19.5	20.4
$15,000 OR MORE	4900	17300	18700	40900	63.6	70.9	72.8	70.8
TOTAL	7700	24400	25700	57800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5400	4400	2600	12400	27.3	13.4	9.5	15.5
$6,000-$14,999	5100	7000	5600	17700	25.8	21.3	20.4	22.1
$15,000 OR MORE	9300	21500	19200	50000	47.0	65.3	70.1	62.4
TOTAL	19800	32900	27400	80100	100.0	100.0	100.0	100.0

TABLE B- 45 MACON, GA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	900	2100	500	3500	34.6	31.8	20.0	29.9
$6,000-$14,999	600	2100	400	3100	23.1	31.8	16.0	26.5
$15,000 OR MORE	1100	2400	1600	5100	42.3	36.4	64.0	43.6
TOTAL	2600	6600	2500	11700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	1000	900	1900	0.0	18.2	11.8	13.9
$6,000-$14,999	300	2500	2400	5200	50.0	45.5	31.6	38.0
$15,000 OR MORE	300	2000	4300	6600	50.0	36.4	56.6	48.2
TOTAL	600	5500	7600	13700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	900	3100	1400	5400	28.1	25.6	13.9	21.3
$6,000-$14,999	900	4600	2800	8300	28.1	38.0	27.7	32.7
$15,000 OR MORE	1400	4400	5900	11700	43.8	36.4	58.4	46.1
TOTAL	3200	12100	10100	25400	100.0	100.0	100.0	100.0

TABLE B- 46 MADISON, WIS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	500	500	1000	0.0	12.5	13.5	9.7
$6,000-$14,999	1000	900	700	2600	38.5	22.5	18.9	25.2
$15,000 OR MORE	1600	2600	2500	6700	61.5	65.0	67.6	65.0
TOTAL	2600	4000	3700	10300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	100	0	600	700	4.5	0.0	8.5	5.4
$6,000-$14,999	700	600	1300	2600	31.8	16.2	18.3	20.0
$15,000 OR MORE	1400	3100	5200	9700	63.6	83.8	73.2	74.6
TOTAL	2200	3700	7100	13000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	100	500	1100	1700	2.1	6.5	10.2	7.3
$6,000-$14,999	1700	1500	2000	5200	35.4	19.5	18.5	22.3
$15,000 OR MORE	3000	5700	7700	16400	62.5	74.0	71.3	70.4
TOTAL	4800	7700	10800	23300	100.0	100.0	100.0	100.0

TABLE B- 47 MCALLEN-PHARR-EDINBURG, TEX.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	800	900	900	2600	34.8	16.1	13.8	18.1
$6,000-$14,999	1400	3400	1700	6500	60.9	60.7	26.2	45.1
$15,000 OR MORE	100	1300	3900	5300	4.3	23.2	60.0	36.8
TOTAL	2300	5600	6500	14400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	900	1600	3400	5900	23.1	25.8	30.9	28.0
$6,000-$14,999	2500	3300	3600	9400	64.1	53.2	32.7	44.5
$15,000 OR MORE	500	1300	4000	5800	12.8	21.0	36.4	27.5
TOTAL	3900	6200	11000	21100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1700	2500	4300	8500	27.4	21.2	24.6	23.9
$6,000-$14,999	3900	6700	5300	15900	62.9	56.8	30.3	44.8
$15,000 OR MORE	600	2600	7900	11100	9.7	22.0	45.1	31.3
TOTAL	6200	11800	17500	35500	100.0	100.0	100.0	100.0

TABLE B- 48 MEMPHIS, TENN.-ARK.-MISS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4200	5900	3900	14000	38.5	17.5	22.7	22.7
$6,000-$14,999	3000	11300	4500	18800	27.5	33.5	26.2	30.4
$15,000 OR MORE	3700	16500	8800	29000	33.9	49.0	51.2	46.9
TOTAL	10900	33700	17200	61800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1300	1700	500	3500	40.6	22.1	3.0	12.7
$6,000-$14,999	600	3100	2600	6300	18.8	40.3	15.7	22.9
$15,000 OR MORE	1300	2900	13500	17700	40.6	37.7	81.3	64.4
TOTAL	3200	7700	16600	27500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5500	7600	4400	17500	39.0	18.4	13.0	19.6
$6,000-$14,999	3600	14400	7100	25100	25.5	34.8	21.0	28.1
$15,000 OR MORE	5000	19400	22300	46700	35.5	46.9	66.0	52.3
TOTAL	14100	41400	33800	89300	100.0	100.0	100.0	100.0

TABLE B- 49 MOBILE, ALA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	1800	500	2900	15.4	20.2	9.6	16.1
$6,000-$14,999	1700	2600	1200	5500	43.6	29.2	23.1	30.6
$15,000 OR MORE	1600	4500	3500	9600	41.0	50.6	67.3	53.3
TOTAL	3900	8900	5200	18000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	900	2500	3800	13.3	12.0	15.5	14.3
$6,000-$14,999	1100	3400	5400	9900	36.7	45.3	33.5	37.2
$15,000 OR MORE	1500	3200	8200	12900	50.0	42.7	50.9	48.5
TOTAL	3000	7500	16100	26600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	2700	3000	6700	14.5	16.5	14.1	15.0
$6,000-$14,999	2800	6000	6600	15400	40.6	36.6	31.0	34.5
$15,000 OR MORE	3100	7700	11700	22500	44.9	47.0	54.9	50.4
TOTAL	6900	16400	21300	44600	100.0	100.0	100.0	100.0

TABLE B- 50 MODESTO, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	400	300	500	1200	28.6	7.0	10.6	11.5
$6,000-$14,999	300	1100	700	2100	21.4	25.6	14.9	20.2
$15,000 OR MORE	700	2900	3500	7100	50.0	67.4	74.5	68.3
TOTAL	1400	4300	4700	10400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	1000	700	2500	19.0	23.3	11.3	17.0
$6,000-$14,999	1300	900	1200	3400	31.0	20.9	19.4	23.1
$15,000 OR MORE	2100	2400	4300	8800	50.0	55.8	69.4	59.9
TOTAL	4200	4300	6200	14700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1200	1300	1200	3700	21.4	15.1	11.0	14.7
$6,000-$14,999	1600	2000	1900	5500	28.6	23.3	17.4	21.9
$15,000 OR MORE	2800	5300	7800	15900	50.0	61.6	71.6	63.3
TOTAL	5600	8600	10900	25100	100.0	100.0	100.0	100.0

TABLE B- 51 NASHVILLE-DAVIDSON, TENN.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1300	3700	1300	6300	18.1	20.8	11.8	17.5
$6,000-$14,999	2100	3800	3000	8900	29.2	21.3	27.3	24.7
$15,000 OR MORE	3800	10300	6700	20800	52.8	57.9	60.9	57.8
TOTAL	7200	17800	11000	36000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	1200	1400	3400	14.8	10.3	7.2	9.3
$6,000-$14,999	1700	3700	5300	10700	31.5	31.6	27.3	29.3
$15,000 OR MORE	2900	6800	12700	22400	53.7	58.1	65.5	61.4
TOTAL	5400	11700	19400	36500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2100	4900	2700	9700	16.7	16.6	8.9	13.4
$6,000-$14,999	3800	7500	8300	19600	30.2	25.4	27.3	27.0
$15,000 OR MORE	6700	17100	19400	43200	53.2	58.0	63.8	59.6
TOTAL	12600	29500	30400	72500	100.0	100.0	100.0	100.0

TABLE B- 52 NEW BRUNSWICK-PERTH AMBOY-SAYREV

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	900	0	1500	14.6	23.7	0.0	18.3
$6,000-$14,999	1300	400	100	1800	31.7	10.5	33.3	22.0
$15,000 OR MORE	2200	2500	200	4900	53.7	65.8	66.7	59.8
TOTAL	4100	3800	300	8200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	800	0	1100	5.3	4.2	0.0	3.2
$6,000-$14,999	400	2100	800	3300	7.0	10.9	8.2	9.5
$15,000 OR MORE	5000	16300	8900	30200	87.7	84.9	91.8	87.3
TOTAL	5700	19200	9700	34600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	900	1700	0	2600	9.2	7.4	0.0	6.1
$6,000-$14,999	1700	2500	900	5100	17.3	10.9	9.0	11.9
$15,000 OR MORE	7200	18800	9100	35100	73.5	81.7	91.0	82.0
TOTAL	9800	23000	10000	42800	100.0	100.0	100.0	100.0

TABLE B- 53 NEW HAVEN-WEST HAVEN, CONN.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2700	1200	200	4100	27.3	32.4	14.3	27.3
$6,000-$14,999	2700	300	300	3300	27.3	8.1	21.4	22.0
$15,000 OR MORE	4500	2200	900	7600	45.5	59.5	64.3	50.7
TOTAL	9900	3700	1400	15000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	300	300	900	5.3	4.3	5.9	5.1
$6,000-$14,999	1400	1000	600	3000	24.6	14.3	11.8	16.9
$15,000 OR MORE	4000	5700	4200	13900	70.2	81.4	82.4	78.1
TOTAL	5700	7000	5100	17800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3000	1500	500	5000	19.2	14.0	7.7	15.2
$6,000-$14,999	4100	1300	900	6300	26.3	12.1	13.8	19.2
$15,000 OR MORE	8500	7900	5100	21500	54.5	73.8	78.5	65.5
TOTAL	15600	10700	6500	32800	100.0	100.0	100.0	100.0

TABLE B- 54 NORFOLK-VIRGINIA BEACH-PORTSMOUT

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
URBAN STATUS/ FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4100	4600	3100	11800	25.6	18.3	16.1	19.5
$6,000-$14,999	6100	8800	5700	20600	38.1	34.9	29.7	34.1
$15,000 OR MORE	5800	11800	10400	28000	36.2	46.8	54.2	46.4
TOTAL	16000	25200	19200	60400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	1000	200	1500	9.7	18.9	2.8	9.7
$6,000-$14,999	1500	2100	2400	6000	48.4	39.6	33.8	38.7
$15,000 OR MORE	1300	2200	4500	8000	41.9	41.5	63.4	51.6
TOTAL	3100	5300	7100	15500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4400	5600	3300	13300	23.0	18.4	12.5	17.5
$6,000-$14,999	7600	10900	8100	26600	39.8	35.7	30.8	35.0
$15,000 OR MORE	7100	14000	14900	36000	37.2	45.9	56.7	47.4
TOTAL	19100	30500	26300	75900	100.0	100.0	100.0	100.0

TABLE B- 55 OKLAHOMA CITY, OKLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1600	2500	1100	5200	20.5	14.1	9.1	13.8
$6,000-$14,999	2000	5100	2500	9600	25.6	28.8	20.7	25.5
$15,000 OR MORE	4200	10100	8500	22800	53.8	57.1	70.2	60.6
TOTAL	7800	17700	12100	37600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1000	1100	1700	3800	18.9	7.7	8.6	9.7
$6,000-$14,999	2400	5000	4200	11600	45.3	35.2	21.2	29.5
$15,000 OR MORE	1900	8100	13900	23900	35.8	57.0	70.2	60.8
TOTAL	5300	14200	19800	39300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2600	3600	2800	9000	19.8	11.3	8.8	11.7
$6,000-$14,999	4400	10100	6700	21200	33.6	31.7	21.0	27.6
$15,000 OR MORE	6100	18200	22400	46700	46.6	57.1	70.2	60.7
TOTAL	13100	31900	31900	76900	100.0	100.0	100.0	100.0

TABLE B- 56 OMAHA, NEBR.-IOWA

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1300	900	0	2200	13.7	9.0	0.0	10.0
$6,000-$14,999	2300	1800	600	4700	24.2	18.0	23.1	21.3
$15,000 OR MORE	5900	7300	2000	15200	62.1	73.0	76.9	68.8
TOTAL	9500	10000	2600	22100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	600	1700	2700	11.1	7.1	10.9	9.7
$6,000-$14,999	1400	2400	2700	6500	38.9	28.2	17.3	23.5
$15,000 OR MORE	1800	5500	11200	18500	50.0	64.7	71.8	66.8
TOTAL	3600	8500	15600	27700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1700	1500	1700	4900	13.0	8.1	9.3	9.8
$6,000-$14,999	3700	4200	3300	11200	28.2	22.7	18.1	22.5
$15,000 OR MORE	7700	12800	13200	33700	58.8	69.2	72.5	67.7
TOTAL	13100	18500	18200	49800	100.0	100.0	100.0	100.0

TABLE B- 57 ORLANDO, FLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	900	1600	300	2800	69.2	23.5	11.5	26.2
$6,000-$14,999	100	1800	500	2400	7.7	26.5	19.2	22.4
$15,000 OR MORE	300	3400	1800	5500	23.1	50.0	69.2	51.4
TOTAL	1300	6800	2600	10700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1200	1500	2300	5000	31.6	9.4	9.5	11.4
$6,000-$14,999	1200	6200	7000	14400	31.6	38.7	29.0	32.8
$15,000 OR MORE	1400	8300	14800	24500	36.8	51.9	61.4	55.8
TOTAL	3800	16000	24100	43900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2100	3100	2600	7800	41.2	13.6	9.7	14.3
$6,000-$14,999	1300	8000	7500	16800	25.5	35.1	28.1	30.8
$15,000 OR MORE	1700	11700	16600	30000	33.3	51.3	62.2	54.9
TOTAL	5100	22800	26700	54600	100.0	100.0	100.0	100.0

TABLE B- 58 OXNARD-SIMI VALLEY-VENTURA, CALI

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	0	1900	600	2500	0.0	13.6	6.1	9.9
$6,000-$14,999	400	4400	1400	6200	28.6	31.4	14.3	24.6
$15,000 OR MORE	1000	7700	7800	16500	71.4	55.0	79.6	65.5
TOTAL	1400	14000	9800	25200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	100	700	1200	2000	2.9	6.9	11.9	8.4
$6,000-$14,999	1300	1700	1300	4300	38.2	16.7	12.9	18.1
$15,000 OR MORE	2000	7800	7600	17400	58.8	76.5	75.2	73.4
TOTAL	3400	10200	10100	23700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	100	2600	1800	4500	2.1	10.7	9.0	9.2
$6,000-$14,999	1700	6100	2700	10500	35.4	25.2	13.6	21.5
$15,000 OR MORE	3000	15500	15400	33900	62.5	64.0	77.4	69.3
TOTAL	4800	24200	19900	48900	100.0	100.0	100.0	100.0

TABLE B- 59 PATERSON-CLIFTON-PASSAIC, N.J.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4400	2200	0	6600	25.9	40.0	0.0	28.2
$6,000-$14,999	4600	800	600	6000	27.1	14.5	66.7	25.6
$15,000 OR MORE	8000	2500	300	10800	47.1	45.5	33.3	46.2
TOTAL	17000	5500	900	23400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	100	200	100	400	2.8	3.6	4.5	3.5
$6,000-$14,999	900	200	0	1100	25.0	3.6	0.0	9.7
$15,000 OR MORE	2600	5100	2100	9800	72.2	92.7	95.5	86.7
TOTAL	3600	5500	2200	11300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4500	2400	100	7000	21.8	21.8	3.2	20.2
$6,000-$14,999	5500	1000	600	7100	26.7	9.1	19.4	20.5
$15,000 OR MORE	10600	7600	2400	20600	51.5	69.1	77.4	59.4
TOTAL	20600	11000	3100	34700	100.0	100.0	100.0	100.0

TABLE B- 60 PEORIA, ILL.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1900	800	0	2700	35.8	16.0	0.0	21.4
$6,000-$14,999	500	1200	300	2000	9.4	24.0	13.0	15.9
$15,000 OR MORE	2900	3000	2000	7900	54.7	60.0	87.0	62.7
TOTAL	5300	5000	2300	12600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	400	600	1400	5.6	4.0	9.5	6.0
$6,000-$14,999	1500	1100	900	3500	20.8	11.1	14.3	15.0
$15,000 OR MORE	5300	8400	4800	18500	73.6	84.8	76.2	79.1
TOTAL	7200	9900	6300	23400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2300	1200	600	4100	18.4	8.1	7.0	11.4
$6,000-$14,999	2000	2300	1200	5500	16.0	15.4	14.0	15.3
$15,000 OR MORE	8200	11400	6800	26400	65.6	76.5	79.1	73.3
TOTAL	12500	14900	8600	36000	100.0	100.0	100.0	100.0

TABLE B- 61 PROVIDENCE-WARWICK-PAWTUCKET, R.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
URBAN STATUS/ FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	4000	400	600	5000	26.7	8.0	37.5	23.1
$6,000-$14,999	4500	1800	100	6400	30.0	36.0	6.3	29.6
$15,000 OR MORE	6500	2800	900	10200	43.3	56.0	56.3	47.2
TOTAL	15000	5000	1600	21600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	2500	400	500	3400	13.7	3.4	4.5	8.3
$6,000-$14,999	6900	2700	1900	11500	37.7	23.3	17.3	28.1
$15,000 OR MORE	8900	8500	8600	26000	48.6	73.3	78.2	63.6
TOTAL	18300	11600	11000	40900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	6500	800	1100	8400	19.5	4.8	8.7	13.4
$6,000-$14,999	11400	4500	2000	17900	34.2	27.1	15.9	28.6
$15,000 OR MORE	15400	11300	9500	36200	46.2	68.1	75.4	57.9
TOTAL	33300	16600	12600	62500	100.0	100.0	100.0	100.0

TABLE B- 62 RALEIGH-DURHAM, N.C.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	1600	1100	3200	14.3	21.1	20.4	19.4
$6,000-$14,999	1700	2800	1300	5800	48.6	36.8	24.1	35.2
$15,000 OR MORE	1300	3200	3000	7500	37.1	42.1	55.6	45.5
TOTAL	3500	7600	5400	16500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	800	1000	1800	0.0	13.3	6.8	8.0
$6,000-$14,999	400	1300	3000	4700	22.2	21.7	20.4	20.9
$15,000 OR MORE	1400	3900	10700	16000	77.8	65.0	72.8	71.1
TOTAL	1800	6000	14700	22500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	500	2400	2100	5000	9.4	17.6	10.4	12.8
$6,000-$14,999	2100	4100	4300	10500	39.6	30.1	21.4	26.9
$15,000 OR MORE	2700	7100	13700	23500	50.9	52.2	68.2	60.3
TOTAL	5300	13600	20100	39000	100.0	100.0	100.0	100.0

TABLE B- 63 RICHMOND, VA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
URBAN STATUS/ FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1200	2200	800	4200	19.0	31.0	30.8	26.2
$6,000-$14,999	2300	1600	500	4400	36.5	22.5	19.2	27.5
$15,000 OR MORE	2800	3300	1300	7400	44.4	46.5	50.0	46.2
TOTAL	6300	7100	2600	16000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	600	500	1300	8.3	5.8	2.8	4.2
$6,000-$14,999	1200	2500	2700	6400	50.0	24.0	15.0	20.8
$15,000 OR MORE	1000	7300	14800	23100	41.7	70.2	82.2	75.0
TOTAL	2400	10400	18000	30800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1400	2800	1300	5500	16.1	16.0	6.3	11.8
$6,000-$14,999	3500	4100	3200	10800	40.2	23.4	15.5	23.1
$15,000 OR MORE	3800	10600	16100	30500	43.7	60.6	78.2	65.2
TOTAL	8700	17500	20600	46800	100.0	100.0	100.0	100.0

TABLE B- 64 ROANOKE, VA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	400	800	600	1800	18.2	19.0	33.3	22.0
$6,000-$14,999	500	1100	400	2000	22.7	26.2	22.2	24.4
$15,000 OR MORE	1300	2300	800	4400	59.1	54.8	44.4	53.7
TOTAL	2200	4200	1800	8200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	200	200	600	16.7	5.9	4.0	6.3
$6,000-$14,999	600	600	900	2100	50.0	17.6	18.0	21.9
$15,000 OR MORE	400	2600	3900	6900	33.3	76.5	78.0	71.9
TOTAL	1200	3400	5000	9600	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	600	1000	800	2400	17.6	13.2	11.8	13.5
$6,000-$14,999	1100	1700	1300	4100	32.4	22.4	19.1	23.0
$15,000 OR MORE	1700	4900	4700	11300	50.0	64.5	69.1	63.5
TOTAL	3400	7600	6800	17800	100.0	100.0	100.0	100.0

TABLE B- 65 ROCHESTER, N.Y.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	3500	1000	200	4700	20.2	27.8	14.3	21.1
$6,000-$14,999	4500	800	900	6200	26.0	22.2	64.3	27.8
$15,000 OR MORE	9300	1800	300	11400	53.8	50.0	21.4	51.1
TOTAL	17300	3600	1400	22300	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1900	1900	700	4500	10.2	9.8	3.7	7.9
$6,000-$14,999	3200	2600	3400	9200	17.2	13.5	18.1	16.2
$15,000 OR MORE	13500	14800	14700	43000	72.6	76.7	78.2	75.8
TOTAL	18600	19300	18800	56700	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	5400	2900	900	9200	15.0	12.7	4.5	11.6
$6,000-$14,999	7700	3400	4300	15400	21.4	14.8	21.3	19.5
$15,000 OR MORE	22800	16600	15000	54400	63.5	72.5	74.3	68.9
TOTAL	35900	22900	20200	79000	100.0	100.0	100.0	100.0

TABLE B- 66 ROCKFORD, ILL.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	700	300	200	1200	12.5	5.9	10.5	9.5
$6,000-$14,999	800	1200	300	2300	14.3	23.5	15.8	18.3
$15,000 OR MORE	4100	3600	1400	9100	73.2	70.6	73.7	72.2
TOTAL	5600	5100	1900	12600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	400	100	700	7.4	7.1	1.8	5.1
$6,000-$14,999	700	1000	600	2300	25.9	17.9	10.9	16.7
$15,000 OR MORE	1800	4200	4800	10800	66.7	75.0	87.3	78.3
TOTAL	2700	5600	5500	13800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	900	700	300	1900	10.8	6.5	4.1	7.2
$6,000-$14,999	1500	2200	900	4600	18.1	20.6	12.2	17.4
$15,000 OR MORE	5900	7800	6200	19900	71.1	72.9	83.8	75.4
TOTAL	8300	10700	7400	26400	100.0	100.0	100.0	100.0

TABLE B- 67 SALINAS-SEASIDE-MONTEREY, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	NUMBER OF CHILDREN TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	PERCENT TOTAL
IN CENTRAL CITY								
UNDER $6,000	500	1000	700	2200	20.8	14.7	14.9	15.8
$6,000-$14,999	1100	2400	1400	4900	45.8	35.3	29.8	35.3
$15,000 OR MORE	800	3400	2600	6800	33.3	50.0	55.3	48.9
TOTAL	2400	6800	4700	13900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	300	500	700	1500	10.3	9.1	12.1	10.6
$6,000-$14,999	1600	1400	1800	4800	55.2	25.5	31.0	33.8
$15,000 OR MORE	1000	3600	3300	7900	34.5	65.5	56.9	55.6
TOTAL	2900	5500	5800	14200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	800	1500	1400	3700	15.1	12.2	13.3	13.2
$6,000-$14,999	2700	3800	3200	9700	50.9	30.9	30.5	34.5
$15,000 OR MORE	1800	7000	5900	14700	34.0	56.9	56.2	52.3
TOTAL	5300	12300	10500	28100	100.0	100.0	100.0	100.0

TABLE B- 68 SALT LAKE CITY-OGDEN, UTAH

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1500	1700	1000	4200	11.4	22.4	20.4	16.3
$6,000-$14,999	4500	2900	1100	8500	34.1	38.2	22.4	33.1
$15,000 OR MORE	7200	3000	2800	13000	54.5	39.5	57.1	50.6
TOTAL	13200	7600	4900	25700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	400	3800	3400	7600	5.4	10.3	4.8	6.6
$6,000-$14,999	2500	10400	14200	27100	33.8	28.1	20.2	23.6
$15,000 OR MORE	4500	22800	52800	80100	60.8	61.6	75.0	69.8
TOTAL	7400	37000	70400	114800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1900	5500	4400	11800	9.2	12.3	5.8	8.4
$6,000-$14,999	7000	13300	15300	35600	34.0	29.8	20.3	25.3
$15,000 OR MORE	11700	25800	55600	93100	56.8	57.8	73.8	66.3
TOTAL	20600	44600	75300	140500	100.0	100.0	100.0	100.0

TABLE B- 69 SANTA BARBARA-SANTA MARIA-LOMPOC

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	300	700	100	1100	13.0	11.3	4.8	10.4
$6,000-$14,999	800	1400	900	3100	34.8	22.6	42.9	29.2
$15,000 OR MORE	1200	4100	1100	6400	52.2	66.1	52.4	60.4
TOTAL	2300	6200	2100	10600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	400	200	600	0.0	6.1	7.1	5.2
$6,000-$14,999	800	2100	400	3300	38.1	31.8	14.3	28.7
$15,000 OR MORE	1300	4100	2200	7600	61.9	62.1	78.6	66.1
TOTAL	2100	6600	2800	11500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	300	1100	300	1700	6.8	8.6	6.1	7.7
$6,000-$14,999	1600	3500	1300	6400	36.4	27.3	26.5	29.0
$15,000 OR MORE	2500	8200	3300	14000	56.8	64.1	67.3	63.3
TOTAL	4400	12800	4900	22100	100.0	100.0	100.0	100.0

TABLE B- 70 SHREVEPORT, LA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1500	2100	900	4500	33.3	20.6	18.0	22.8
$6,000-$14,999	1600	2600	500	4700	35.6	25.5	10.0	23.9
$15,000 OR MORE	1400	5500	3600	10500	31.1	53.9	72.0	53.3
TOTAL	4500	10200	5000	19700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1000	900	200	2100	32.3	10.8	2.8	11.4
$6,000-$14,999	1200	3300	1900	6400	38.7	39.8	26.8	34.6
$15,000 OR MORE	900	4100	5000	10000	29.0	49.4	70.4	54.1
TOTAL	3100	8300	7100	18500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2500	3000	1100	6600	32.9	16.2	9.1	17.3
$6,000-$14,999	2800	5900	2400	11100	36.8	31.9	19.8	29.1
$15,000 OR MORE	2300	9600	8600	20500	30.3	51.9	71.1	53.7
TOTAL	7600	18500	12100	38200	100.0	100.0	100.0	100.0

TABLE B- 71 SOUTH BEND, IND.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	900	700	300	1900	13.2	15.6	25.0	15.2
$6,000-$14,999	2600	1200	400	4200	38.2	26.7	33.3	33.6
$15,000 OR MORE	3300	2600	500	6400	48.5	57.8	41.7	51.2
TOTAL	6800	4500	1200	12500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	700	200	200	1100	12.1	4.4	4.3	7.3
$6,000-$14,999	2000	1000	800	3800	34.5	22.2	17.0	25.3
$15,000 OR MORE	3100	3300	3700	10100	53.4	73.3	78.7	67.3
TOTAL	5800	4500	4700	15000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1600	900	500	3000	12.7	10.0	8.5	10.9
$6,000-$14,999	4600	2200	1200	8000	36.5	24.4	20.3	29.1
$15,000 OR MORE	6400	5900	4200	16500	50.8	65.6	71.2	60.0
TOTAL	12600	9000	5900	27500	100.0	100.0	100.0	100.0

TABLE B- 72 SPOKANE, WASH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1100	100	700	1900	16.2	2.6	16.3	12.8
$6,000-$14,999	2400	1000	1000	4400	35.3	26.3	23.3	29.5
$15,000 OR MORE	3300	2700	2600	8600	48.5	71.1	60.5	57.7
TOTAL	6800	3800	4300	14900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	400	700	1100	0.0	8.0	6.7	6.1
$6,000-$14,999	700	1100	1800	3600	26.9	22.0	17.1	19.9
$15,000 OR MORE	1900	3500	8000	13400	73.1	70.0	76.2	74.0
TOTAL	2600	5000	10500	18100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1100	500	1400	3000	11.7	5.7	9.5	9.1
$6,000-$14,999	3100	2100	2800	8000	33.0	23.9	18.9	24.2
$15,000 OR MORE	5200	6200	10600	22000	55.3	70.5	71.6	66.7
TOTAL	9400	8800	14800	33000	100.0	100.0	100.0	100.0

TABLE B- 73 SPRINGFIELD-CHICOPEE-HOLYOKE, MA

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2800	500	600	3900	22.6	13.5	19.4	20.3
$6,000-$14,999	3700	1000	1100	5800	29.8	27.0	35.5	30.2
$15,000 OR MORE	5900	2200	1400	9500	47.6	59.5	45.2	49.5
TOTAL	12400	3700	3100	19200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	600	600	400	1600	11.8	11.1	5.9	9.2
$6,000-$14,999	1700	900	900	3500	33.3	16.7	13.2	20.2
$15,000 OR MORE	2800	3900	5500	12200	54.9	72.2	80.9	70.5
TOTAL	5100	5400	6800	17300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3400	1100	1000	5500	19.4	12.1	10.1	15.1
$6,000-$14,999	5400	1900	2000	9300	30.9	20.9	20.2	25.5
$15,000 OR MORE	8700	6100	6900	21700	49.7	67.0	69.7	59.5
TOTAL	17500	9100	9900	36500	100.0	100.0	100.0	100.0

TABLE B- 74 STOCKTON, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	800	1900	1300	4000	27.6	28.8	19.7	24.8
$6,000-$14,999	600	1800	700	3100	20.7	27.3	10.6	19.3
$15,000 OR MORE	1500	2900	4600	9000	51.7	43.9	69.7	55.9
TOTAL	2900	6600	6600	16100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	1100	800	2700	17.4	15.1	11.4	14.3
$6,000-$14,999	1400	2300	2400	6100	30.4	31.5	34.3	32.3
$15,000 OR MORE	2400	3900	3800	10100	52.2	53.4	54.3	53.4
TOTAL	4600	7300	7000	18900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1600	3000	2100	6700	21.3	21.6	15.4	19.1
$6,000-$14,999	2000	4100	3100	9200	26.7	29.5	22.8	26.3
$15,000 OR MORE	3900	6800	8400	19100	52.0	48.9	61.8	54.6
TOTAL	7500	13900	13600	35000	100.0	100.0	100.0	100.0

TABLE B- 75 SYRACUSE, N.Y.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2300	800	0	3100	22.3	27.6	0.0	23.0
$6,000-$14,999	3700	900	100	4700	35.9	31.0	33.3	34.8
$15,000 OR MORE	4300	1200	200	5700	41.7	41.4	66.7	42.2
TOTAL	10300	2900	300	13500	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	1800	400	900	3100	12.5	3.4	6.2	7.7
$6,000-$14,999	3300	1900	2300	7500	22.9	16.4	15.9	18.5
$15,000 OR MORE	9300	9300	11300	29900	64.6	80.2	77.9	73.8
TOTAL	14400	11600	14500	40500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	4100	1200	900	6200	16.6	8.3	6.1	11.5
$6,000-$14,999	7000	2800	2400	12200	28.3	19.3	16.2	22.6
$15,000 OR MORE	13600	10500	11500	35600	55.1	72.4	77.7	65.9
TOTAL	24700	14500	14800	54000	100.0	100.0	100.0	100.0

TABLE B- 76 TACOMA, WASH.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	400	1000	700	2100	5.2	20.8	20.0	13.1
$6,000-$14,999	2200	1700	1200	5100	28.6	35.4	34.3	31.9
$15,000 OR MORE	5100	2100	1600	8800	66.2	43.8	45.7	55.0
TOTAL	7700	4800	3500	16000	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	700	1300	2800	14.5	7.1	7.8	8.7
$6,000-$14,999	1500	4200	4700	10400	27.3	42.4	28.3	32.5
$15,000 OR MORE	3200	5000	10600	18800	58.2	50.5	63.9	58.7
TOTAL	5500	9900	16600	32000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1200	1700	2000	4900	9.1	11.6	10.0	10.2
$6,000-$14,999	3700	5900	5900	15500	28.0	40.1	29.4	32.3
$15,000 OR MORE	8300	7100	12200	27600	62.9	48.3	60.7	57.5
TOTAL	13200	14700	20100	48000	100.0	100.0	100.0	100.0

TABLE B- 77 TUCSON, ARIZ.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	600	2400	1100	4100	27.3	16.9	9.7	14.8
$6,000-$14,999	1000	5200	3600	9800	45.5	36.6	31.9	35.4
$15,000 OR MORE	600	6600	6600	13800	27.3	46.5	58.4	49.8
TOTAL	2200	14200	11300	27700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	400	400	800	0.0	8.7	3.9	5.2
$6,000-$14,999	200	1900	2000	4100	40.0	41.3	19.4	26.6
$15,000 OR MORE	300	2300	7900	10500	60.0	50.0	76.7	68.2
TOTAL	500	4600	10300	15400	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	600	2800	1500	4900	22.2	14.9	6.9	11.4
$6,000-$14,999	1200	7100	5600	13900	44.4	37.8	25.9	32.3
$15,000 OR MORE	900	8900	14500	24300	33.3	47.3	67.1	56.4
TOTAL	2700	18800	21600	43100	100.0	100.0	100.0	100.0

TABLE B- 78 TULSA, OKLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	800	2200	300	3300	13.3	13.7	3.3	10.6
$6,000-$14,999	1700	6300	1700	9700	28.3	39.1	18.7	31.1
$15,000 OR MORE	3500	7600	7100	18200	58.3	47.2	78.0	58.3
TOTAL	6000	16100	9100	31200	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	900	1600	1200	3700	15.3	16.3	6.3	10.6
$6,000-$14,999	2600	3600	3900	10100	44.1	36.7	20.3	28.9
$15,000 OR MORE	2400	4600	14100	21100	40.7	46.9	73.4	60.5
TOTAL	5900	9800	19200	34900	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1700	3800	1500	7000	14.3	14.7	5.3	10.6
$6,000-$14,999	4300	9900	5600	19800	36.1	38.2	19.8	30.0
$15,000 OR MORE	5900	12200	21200	39300	49.6	47.1	74.9	59.5
TOTAL	11900	25900	28300	66100	100.0	100.0	100.0	100.0

TABLE B- 79 UTICA-ROME, N.Y.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1500	600	0	2100	26.3	20.0	0.0	21.9
$6,000-$14,999	2400	1800	200	4400	42.1	60.0	22.2	45.8
$15,000 OR MORE	1800	600	700	3100	31.6	20.0	77.8	32.3
TOTAL	5700	3000	900	9600	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	800	200	100	1100	12.7	3.9	2.1	6.8
$6,000-$14,999	2100	900	1700	4700	33.3	17.6	36.2	29.2
$15,000 OR MORE	3400	4000	2900	10300	54.0	78.4	61.7	64.0
TOTAL	6300	5100	4700	16100	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2300	800	100	3200	19.2	9.9	1.8	12.5
$6,000-$14,999	4500	2700	1900	9100	37.5	33.3	33.9	35.4
$15,000 OR MORE	5200	4600	3600	13400	43.3	56.8	64.3	52.1
TOTAL	12000	8100	5600	25700	100.0	100.0	100.0	100.0

TABLE B- 80 VALLEJO-FAIRFIELD-NAPA, CALIF.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	700	1400	100	2200	16.3	17.3	1.5	11.5
$6,000-$14,999	1500	2500	900	4900	34.9	30.9	13.4	25.7
$15,000 OR MORE	2100	4200	5700	12000	48.8	51.9	85.1	62.8
TOTAL	4300	8100	6700	19100	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	100	300	100	500	11.1	8.1	1.4	4.2
$6,000-$14,999	200	900	500	1600	22.2	24.3	6.9	13.6
$15,000 OR MORE	600	2500	6600	9700	66.7	67.6	91.7	82.2
TOTAL	900	3700	7200	11800	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	800	1700	200	2700	15.4	14.4	1.4	8.7
$6,000-$14,999	1700	3400	1400	6500	32.7	28.8	10.1	21.0
$15,000 OR MORE	2700	6700	12300	21700	51.9	56.8	88.5	70.2
TOTAL	5200	11800	13900	30900	100.0	100.0	100.0	100.0

TABLE B- 81 WATERBURY, CONN.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
URBAN STATUS/ FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	800	500	0	1300	14.0	29.4	0.0	14.8
$6,000-$14,999	1400	300	200	1900	24.6	17.6	14.3	21.6
$15,000 OR MORE	3500	900	1200	5600	61.4	52.9	85.7	63.6
TOTAL	5700	1700	1400	8800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	200	200	300	700	8.0	5.4	7.3	6.8
$6,000-$14,999	400	0	500	900	16.0	0.0	12.2	8.7
$15,000 OR MORE	1900	3500	3300	8700	76.0	94.6	80.5	84.5
TOTAL	2500	3700	4100	10300	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1000	700	300	2000	12.2	13.0	5.5	10.5
$6,000-$14,999	1800	300	700	2800	22.0	5.6	12.7	14.7
$15,000 OR MORE	5400	4400	4500	14300	65.9	81.5	81.8	74.9
TOTAL	8200	5400	5500	19100	100.0	100.0	100.0	100.0

TABLE B- 82 WEST PALM BEACH-BOCA RATON, FLA.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	200	200	400	800	22.2	5.9	19.0	12.5
$6,000-$14,999	200	1000	400	1600	22.2	29.4	19.0	25.0
$15,000 OR MORE	500	2200	1300	4000	55.6	64.7	61.9	62.5
TOTAL	900	3400	2100	6400	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	2400	1700	4100	0.0	18.8	11.3	14.0
$6,000-$14,999	600	3200	2600	6400	46.2	25.0	17.2	21.9
$15,000 OR MORE	700	7200	10800	18700	53.8	56.3	71.5	64.0
TOTAL	1300	12800	15100	29200	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	200	2600	2100	4900	9.1	16.0	12.2	13.8
$6,000-$14,999	800	4200	3000	8000	36.4	25.9	17.4	22.5
$15,000 OR MORE	1200	9400	12100	22700	54.5	58.0	70.3	63.8
TOTAL	2200	16200	17200	35600	100.0	100.0	100.0	100.0

TABLE B- 83 WICHITA, KANS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1700	1800	500	4000	16.3	13.8	11.6	14.4
$6,000-$14,999	3700	4100	400	8200	35.6	31.5	9.3	29.6
$15,000 OR MORE	5000	7100	3400	15500	48.1	54.6	79.1	56.0
TOTAL	10400	13000	4300	27700	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	0	0	200	200	0.0	0.0	3.0	1.4
$6,000-$14,999	800	1900	1600	4300	24.2	46.3	24.2	30.7
$15,000 OR MORE	2500	2200	4800	9500	75.8	53.7	72.7	67.9
TOTAL	3300	4100	6600	14000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	1700	1800	700	4200	12.4	10.5	6.4	10.1
$6,000-$14,999	4500	6000	2000	12500	32.8	35.1	18.3	30.0
$15,000 OR MORE	7500	9300	8200	25000	54.7	54.4	75.2	60.0
TOTAL	13700	17100	10900	41700	100.0	100.0	100.0	100.0

TABLE B- 84 WORCESTER, MASS.

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	1500	600	300	2400	21.4	21.4	30.0	22.2
$6,000-$14,999	2000	600	200	2800	28.6	21.4	20.0	25.9
$15,000 OR MORE	3500	1600	500	5600	50.0	57.1	50.0	51.9
TOTAL	7000	2800	1000	10800	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	500	0	200	700	7.8	0.0	3.4	4.5
$6,000-$14,999	2100	800	1100	4000	32.8	25.0	18.6	25.8
$15,000 OR MORE	3800	2400	4600	10800	59.4	75.0	78.0	69.7
TOTAL	6400	3200	5900	15500	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	2000	600	500	3100	14.9	10.0	7.2	11.8
$6,000-$14,999	4100	1400	1300	6800	30.6	23.3	18.8	25.9
$15,000 OR MORE	7300	4000	5100	16400	54.5	66.7	73.9	62.4
TOTAL	13400	6000	6900	26300	100.0	100.0	100.0	100.0

TABLE B- 85 YOUNGSTOWN-WARREN, OHIO

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, URBAN STATUS AND AGE OF HOUSING, 1980

URBAN STATUS/ FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
IN CENTRAL CITY								
UNDER $6,000	2400	1900	0	4300	28.2	30.6	0.0	28.9
$6,000-$14,999	2200	1300	0	3500	25.9	21.0	0.0	23.5
$15,000 OR MORE	3900	3000	200	7100	45.9	48.4	100.0	47.7
TOTAL	8500	6200	200	14900	100.0	100.0	100.0	100.0
NOT IN CENTRAL CITY								
UNDER $6,000	700	200	400	1300	7.4	2.5	3.2	4.3
$6,000-$14,999	2900	1000	1900	5800	30.5	12.3	15.3	19.3
$15,000 OR MORE	5900	6900	10100	22900	62.1	85.2	81.5	76.3
TOTAL	9500	8100	12400	30000	100.0	100.0	100.0	100.0
TOTAL SMSA								
UNDER $6,000	3100	2100	400	5600	17.2	14.7	3.2	12.5
$6,000-$14,999	5100	2300	1900	9300	28.3	16.1	15.1	20.7
$15,000 OR MORE	9800	9900	10300	30000	54.4	69.2	81.7	66.8
TOTAL	18000	14300	12600	44900	100.0	100.0	100.0	100.0

APPENDIX C

TABLES OF INDIVIDUAL AND MERGED SMSAs WITH POPULATIONS OF LESS THAN 500,000 SHOWING NUMBERS OF YOUNG CHILDREN BY THE AGE OF THEIR HOUSING AND FAMILY INCOME

The tables in this Appendix include 195 SMSAs with total populations under 1 million where the Census data tape did not show the residential categories "In Central City" and "Not In Central City." They are shown in alphabetical order by the city that gives its name to the SMSA.

The data came from tapes of 1980 U.S. Census enumerations and cover children aged 6 months to 5 years of all races. Distribution by family income and age of residential unit is given.

Binghampton, NY-PA, is shown without the population for Tioga County. This population is combined with Elmira, NY.

The following SMSAs had been combined on the data tape, and single tables only can be shown for the pairs:

Bangor, ME, and Lewiston-Auburn, ME
Bismarck, ND, and Grand Forks, ND
Bloomington, IN, and Owensboro, KY
Bristol, CT, and Meriden, CT
Bryan-College Station, TX, and Sherman-Denison, TX
Casper, WY, and Great Falls, MT
Dubuque, IA, and Iowa City, IA
El Paso, TX, and Las Cruces, NM
Fitchburg-Leominster, MA, and Pittsfield, MA
Ft. Walton Beach, FL, and Panama City, FL
Greensboro, Winston Salem, and High Point, NC, and Burlington, NC
La Crosse, WI, and Rochester, MN
Laredo, TX, and Victoria, TX
Lawton, OK, and Enid, OK
Little Rock-North Little Rock, AR, and Pine Bluff, AR
Midland, TX, and San Angelo, TX
Topeka, KS, and Lawrence, KS

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 1 ABILENE, TEX.								
UNDER $6,000	100	400	400	900	3.1	7.5	18.2	8.4
$6,000-$14,999	1400	1700	500	3600	43.8	32.1	22.7	33.6
$15,000 OR MORE	1700	3200	1300	6200	53.1	60.4	59.1	57.9
TOTAL	3200	5300	2200	10700	100.0	100.0	100.0	100.0
TABLE C- 2 ALBANY, GA.								
UNDER $6,000	900	1900	1200	4000	60.0	39.6	21.8	33.9
$6,000-$14,999	100	500	1200	1800	6.7	10.4	21.8	15.3
$15,000 OR MORE	500	2400	3100	6000	33.3	50.0	56.4	50.8
TOTAL	1500	4800	5500	11800	100.0	100.0	100.0	100.0
TABLE C- 3 ALEXANDRIA, LA.								
UNDER $6,000	1200	1100	700	3000	32.4	14.1	11.5	17.0
$6,000-$14,999	1500	3600	2300	7400	40.5	46.2	37.7	42.0
$15,000 OR MORE	1000	3100	3100	7200	27.0	39.7	50.8	40.9
TOTAL	3700	7800	6100	17600	100.0	100.0	100.0	100.0
TABLE C- 4 ALTOONA, PA.								
UNDER $6,000	300	400	200	900	6.0	19.0	4.9	8.0
$6,000-$14,999	1500	700	1500	3700	30.0	33.3	36.6	33.0
$15,000 OR MORE	3200	1000	2400	6600	64.0	47.6	58.5	58.9
TOTAL	5000	2100	4100	11200	100.0	100.0	100.0	100.0
TABLE C- 5 AMARILLO, TEX.								
UNDER $6,000	800	1100	600	2500	20.5	12.5	11.3	13.9
$6,000-$14,999	1900	3000	1100	6000	48.7	34.1	20.8	33.3
$15,000 OR MORE	1200	4700	3600	9500	30.8	53.4	67.9	52.8
TOTAL	3900	8800	5300	18000	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 6 ANCHORAGE, ALASKA								
UNDER $6,000	200	700	600	1500	14.3	8.1	5.9	7.5
$6,000-$14,999	700	2900	1000	4600	50.0	33.7	9.9	22.9
$15,000 OR MORE	500	5000	8500	14000	35.7	58.1	84.2	69.7
TOTAL	1400	8600	10100	20100	100.0	100.0	100.0	100.0
TABLE C- 7 ANDERSON, IND.								
UNDER $6,000	1600	1300	100	3000	25.8	25.5	5.6	22.9
$6,000-$14,999	1300	900	0	2200	21.0	17.6	0.0	16.8
$15,000 OR MORE	3300	2900	1700	7900	53.2	56.9	94.4	60.3
TOTAL	6200	5100	1800	13100	100.0	100.0	100.0	100.0
TABLE C- 8 ANDERSON, S.C.								
UNDER $6,000	800	400	300	1500	28.6	11.1	7.5	14.4
$6,000-$14,999	800	1000	900	2700	28.6	27.8	22.5	26.0
$15,000 OR MORE	1200	2200	2800	6200	42.9	61.1	70.0	59.6
TOTAL	2800	3600	4000	10400	100.0	100.0	100.0	100.0
TABLE C- 9 ANNISTON, ALA.								
UNDER $6,000	900	400	200	1500	29.0	11.8	5.4	14.7
$6,000-$14,999	900	2100	1400	4400	29.0	61.8	37.8	43.1
$15,000 OR MORE	1300	900	2100	4300	41.9	26.5	56.8	42.2
TOTAL	3100	3400	3700	10200	100.0	100.0	100.0	100.0
TABLE C- 10 ASHEVILLE, N.C.								
UNDER $6,000	100	400	900	1400	4.2	9.5	16.7	11.7
$6,000-$14,999	1200	1200	900	3300	50.0	28.6	16.7	27.5
$15,000 OR MORE	1100	2600	3600	7300	45.8	61.9	66.7	60.8
TOTAL	2400	4200	5400	12000	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 11 ATHENS, GA.								
UNDER $6,000	300	500	700	1500	17.6	12.2	13.7	13.8
$6,000-$14,999	400	1300	2300	4000	23.5	31.7	45.1	36.7
$15,000 OR MORE	1000	2300	2100	5400	58.8	56.1	41.2	49.5
TOTAL	1700	4100	5100	10900	100.0	100.0	100.0	100.0
TABLE C- 12 ATLANTIC CITY, N.J.								
UNDER $6,000	500	500	300	1300	12.2	12.2	5.3	9.4
$6,000-$14,999	1500	900	700	3100	36.6	22.0	12.3	22.3
$15,000 OR MORE	2100	2700	4700	9500	51.2	65.9	82.5	68.3
TOTAL	4100	4100	5700	13900	100.0	100.0	100.0	100.0
TABLE C- 13 AUGUSTA, GA.-S.C.								
UNDER $6,000	800	1700	800	3300	24.2	13.1	5.8	11.0
$6,000-$14,999	1200	5300	5300	11800	36.4	40.8	38.7	39.3
$15,000 OR MORE	1300	6000	7600	14900	39.4	46.2	55.5	49.7
TOTAL	3300	13000	13700	30000	100.0	100.0	100.0	100.0
TABLE C- 14 BANGOR, ME AND LEWISTON-AUBURN, ME								
UNDER $6,000	800	300	200	1300	12.7	11.1	7.7	11.2
$6,000-$14,999	2000	1200	800	4000	31.7	44.4	30.8	34.5
$15,000 OR MORE	3500	1200	1600	6300	55.6	44.4	61.5	54.3
TOTAL	6300	2700	2600	11600	100.0	100.0	100.0	100.0
TABLE C- 15 BATTLE CREEK, MICH.								
UNDER $6,000	1300	1100	400	2800	17.1	17.5	14.8	16.9
$6,000-$14,999	2600	1600	600	4800	34.2	25.4	22.2	28.9
$15,000 OR MORE	3700	3600	1700	9000	48.7	57.1	63.0	54.2
TOTAL	7600	6300	2700	16600	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 16 BAY CITY, MICH.								
UNDER $6,000	300	600	700	1600	11.1	17.6	15.6	15.1
$6,000-$14,999	800	600	500	1900	29.6	17.6	11.1	17.9
$15,000 OR MORE	1600	2200	3300	7100	59.3	64.7	73.3	67.0
TOTAL	2700	3400	4500	10600	100.0	100.0	100.0	100.0
TABLE C- 17 BELLINGHAM, WASH.								
UNDER $6,000	100	0	500	600	3.1	0.0	13.9	7.1
$6,000-$14,999	1000	200	700	1900	31.3	12.5	19.4	22.6
$15,000 OR MORE	2100	1400	2400	5900	65.6	87.5	66.7	70.2
TOTAL	3200	1600	3600	8400	100.0	100.0	100.0	100.0
TABLE C- 18 BENTON HARBOR, MICH.								
UNDER $6,000	700	700	800	2200	13.7	10.8	21.6	14.4
$6,000-$14,999	2100	2500	1200	5800	41.2	38.5	32.4	37.9
$15,000 OR MORE	2300	3300	1700	7300	45.1	50.8	45.9	47.7
TOTAL	5100	6500	3700	15300	100.0	100.0	100.0	100.0
TABLE C- 19 BILLINGS, MONT.								
UNDER $6,000	100	500	500	1100	3.7	16.7	9.8	10.2
$6,000-$14,999	1200	900	700	2800	44.4	30.0	13.7	25.9
$15,000 OR MORE	1400	1600	3900	6900	51.9	53.3	76.5	63.9
TOTAL	2700	3000	5100	10800	100.0	100.0	100.0	100.0
TABLE C- 20 BILOXI-GULFPORT, MISS.								
UNDER $6,000	700	1100	500	2300	22.6	12.9	7.7	12.7
$6,000-$14,999	1300	3900	2700	7900	41.9	45.9	41.5	43.6
$15,000 OR MORE	1100	3500	3300	7900	35.5	41.2	50.8	43.6
TOTAL	3100	8500	6500	18100	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C- 21 BINGHAMTON, N.Y.-PA.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1300	200	100	1600	11.9	3.6	2.6	7.9
$6,000-$14,999	3600	1500	1400	6500	33.0	27.3	35.9	32.0
$15,000 OR MORE	6000	3800	2400	12200	55.0	69.1	61.5	60.1
TOTAL	10900	5500	3900	20300	100.0	100.0	100.0	100.0

TABLE C- 22 BISMARCK, ND AND GRAND FORKS, ND-MN

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	200	800	1300	5.9	3.3	9.1	6.5
$6,000-$14,999	2600	2400	2500	7500	51.0	40.0	28.4	37.7
$15,000 OR MORE	2200	3400	5500	11100	43.1	56.7	62.5	55.8
TOTAL	5100	6000	8800	19900	100.0	100.0	100.0	100.0

TABLE C- 23 BLOOMINGTON, IN AND OWENSBORO, KY

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	800	700	700	2200	21.6	12.3	11.1	14.0
$6,000-$14,999	1100	1600	1600	4300	29.7	28.1	25.4	27.4
$15,000 OR MORE	1800	3400	4000	9200	48.6	59.6	63.5	58.6
TOTAL	3700	5700	6300	15700	100.0	100.0	100.0	100.0

TABLE C- 24 BLOOMINGTON-NORMAL, ILL.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	400	100	200	700	9.3	8.3	5.1	7.4
$6,000-$14,999	1000	200	900	2100	23.3	16.7	23.1	22.3
$15,000 OR MORE	2900	900	2800	6600	67.4	75.0	71.8	70.2
TOTAL	4300	1200	3900	9400	100.0	100.0	100.0	100.0

TABLE C- 25 BOISE CITY, IDAHO

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	200	600	1100	12.0	7.4	5.4	6.7
$6,000-$14,999	700	1200	3700	5600	28.0	44.4	33.0	34.1
$15,000 OR MORE	1500	1300	6900	9700	60.0	48.1	61.6	59.1
TOTAL	2500	2700	11200	16400	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 26 BRADENTON, FLA.								
UNDER $6,000	100	500	700	1300	5.9	16.7	14.6	13.7
$6,000-$14,999	1100	400	1500	3000	64.7	13.3	31.3	31.6
$15,000 OR MORE	500	2100	2600	5200	29.4	70.0	54.2	54.7
TOTAL	1700	3000	4800	9500	100.0	100.0	100.0	100.0
TABLE C- 27 BREMERTON, WASH.								
UNDER $6,000	400	300	700	1400	15.4	13.0	7.9	10.1
$6,000-$14,999	700	800	1100	2600	26.9	34.8	12.4	18.8
$15,000 OR MORE	1500	1200	7100	9800	57.7	52.2	79.8	71.0
TOTAL	2600	2300	8900	13800	100.0	100.0	100.0	100.0
TABLE C- 28 BRISTOL, CT AND MERIDEN, CT								
UNDER $6,000	1000	200	100	1300	18.5	4.9	4.5	11.1
$6,000-$14,999	900	1100	300	2300	16.7	26.8	13.6	19.7
$15,000 OR MORE	3500	2800	1800	8100	64.8	68.3	81.8	69.2
TOTAL	5400	4100	2200	11700	100.0	100.0	100.0	100.0
TABLE C- 29 BROCKTON, MASS.								
UNDER $6,000	800	300	200	1300	18.2	7.3	5.6	10.7
$6,000-$14,999	1600	600	400	2600	36.4	14.6	11.1	21.5
$15,000 OR MORE	2000	3200	3000	8200	45.5	78.0	83.3	67.8
TOTAL	4400	4100	3600	12100	100.0	100.0	100.0	100.0
TABLE C- 30 BROWNSVILLE-HARLINGEN-SAN BENITO, TEX.								
UNDER $6,000	900	1600	1900	4400	19.1	14.5	17.4	16.5
$6,000-$14,999	2200	5000	4900	12100	46.8	45.5	45.0	45.5
$15,000 OR MORE	1600	4400	4100	10100	34.0	40.0	37.6	38.0
TOTAL	4700	11000	10900	26600	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 31 BRYAN-COLLEGE STATION, TX AND SHERMAN-DENNISON, TX								
UNDER $6,000	0	300	400	700	0.0	5.1	7.0	5.0
$6,000-$14,999	1100	2700	1100	4900	45.8	45.8	19.3	35.0
$15,000 OR MORE	1300	2900	4200	8400	54.2	49.2	73.7	60.0
TOTAL	2400	5900	5700	14000	100.0	100.0	100.0	100.0
TABLE C- 32 BURLINGTON, VT.								
UNDER $6,000	0	800	0	800	0.0	25.8	0.0	8.3
$6,000-$14,999	800	600	1200	2600	40.0	19.4	26.7	27.1
$15,000 OR MORE	1200	1700	3300	6200	60.0	54.8	73.3	64.6
TOTAL	2000	3100	4500	9600	100.0	100.0	100.0	100.0
TABLE C- 33 CANTON, OHIO								
UNDER $6,000	2500	900	600	4000	17.4	8.3	6.4	11.5
$6,000-$14,999	3600	2600	1200	7400	25.0	23.9	12.8	21.3
$15,000 OR MORE	8300	7400	7600	23300	57.6	67.9	80.9	67.1
TOTAL	14400	10900	9400	34700	100.0	100.0	100.0	100.0
TABLE C- 34 CASPER, WY AND GREAT FALLS, MT								
UNDER $6,000	0	300	300	600	0.0	7.0	3.2	3.5
$6,000-$14,999	2000	1000	2100	5100	57.1	23.3	22.6	29.8
$15,000 OR MORE	1500	3000	6900	11400	42.9	69.8	74.2	66.7
TOTAL	3500	4300	9300	17100	100.0	100.0	100.0	100.0
TABLE C- 35 CEDAR RAPIDS, IOWA								
UNDER $6,000	600	100	200	900	13.6	1.9	4.2	6.3
$6,000-$14,999	600	700	800	2100	13.6	13.5	16.7	14.6
$15,000 OR MORE	3200	4400	3800	11400	72.7	84.6	79.2	79.2
TOTAL	4400	5200	4800	14400	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 36 CHAMPAIGN-URBANA-RANTOUL, ILL.								
UNDER $6,000	200	1100	400	1700	7.4	17.5	10.3	13.2
$6,000-$14,999	500	1300	600	2400	18.5	20.6	15.4	18.6
$15,000 OR MORE	2000	3900	2900	8800	74.1	61.9	74.4	68.2
TOTAL	2700	6300	3900	12900	100.0	100.0	100.0	100.0
TABLE C- 37 CHARLESTON, W. VA.								
UNDER $6,000	800	400	800	2000	12.5	5.6	10.5	9.5
$6,000-$14,999	1600	2300	1700	5600	25.0	32.4	22.4	26.5
$15,000 OR MORE	4000	4400	5100	13500	62.5	62.0	67.1	64.0
TOTAL	6400	7100	7600	21100	100.0	100.0	100.0	100.0
TABLE C- 38 CHARLOTTESVILLE, VA.								
UNDER $6,000	200	0	400	600	22.2	0.0	11.1	9.2
$6,000-$14,999	100	1000	1500	2600	11.1	50.0	41.7	40.0
$15,000 OR MORE	600	1000	1700	3300	66.7	50.0	47.2	50.8
TOTAL	900	2000	3600	6500	100.0	100.0	100.0	100.0
TABLE C- 39 CHICO, CALIF.								
UNDER $6,000	400	600	300	1300	22.2	13.6	7.1	12.5
$6,000-$14,999	600	1500	700	2800	33.3	34.1	16.7	26.9
$15,000 OR MORE	800	2300	3200	6300	44.4	52.3	76.2	60.6
TOTAL	1800	4400	4200	10400	100.0	100.0	100.0	100.0
TABLE C- 40 CLARKSVILLE-HOPKINSVILLE, TENN.-KY.								
UNDER $6,000	100	400	800	1300	7.7	7.4	10.5	9.1
$6,000-$14,999	1000	3500	3300	7800	76.9	64.8	43.4	54.5
$15,000 OR MORE	200	1500	3500	5200	15.4	27.8	46.1	36.4
TOTAL	1300	5400	7600	14300	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C- 41 COLUMBIA, MO.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	0	800	200	1000	0.0	23.5	4.1	11.2
$6,000-$14,999	200	600	1700	2500	33.3	17.6	34.7	28.1
$15,000 OR MORE	400	2000	3000	5400	66.7	58.8	61.2	60.7
TOTAL	600	3400	4900	8900	100.0	100.0	100.0	100.0

TABLE C- 42 COLUMBUS, GA.-ALA.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	800	2600	300	3700	25.0	20.8	4.8	16.8
$6,000-$14,999	1600	4900	1900	8400	50.0	39.2	30.2	38.2
$15,000 OR MORE	800	5000	4100	9900	25.0	40.0	65.1	45.0
TOTAL	3200	12500	6300	22000	100.0	100.0	100.0	100.0

TABLE C- 43 CORPUS CHRISTI, TEX.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	2200	2200	900	5300	22.2	14.1	8.8	14.8
$6,000-$14,999	3500	5400	3400	12300	35.4	34.6	33.3	34.5
$15,000 OR MORE	4200	8000	5900	18100	42.4	51.3	57.8	50.7
TOTAL	9900	15600	10200	35700	100.0	100.0	100.0	100.0

TABLE C- 44 CUMBERLAND, MD.-W. VA.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1100	200	0	1300	26.2	10.0	0.0	17.8
$6,000-$14,999	1200	900	400	2500	28.6	45.0	36.4	34.2
$15,000 OR MORE	1900	900	700	3500	45.2	45.0	63.6	47.9
TOTAL	4200	2000	1100	7300	100.0	100.0	100.0	100.0

TABLE C- 45 DANBURY, CONN.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	100	100	200	400	3.4	2.8	4.1	3.5
$6,000-$14,999	1400	400	100	1900	48.3	11.1	2.0	16.7
$15,000 OR MORE	1400	3100	4600	9100	48.3	86.1	93.9	79.8
TOTAL	2900	3600	4900	11400	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 46 DANVILLE, VA.								
UNDER $6,000	600	200	300	1100	20.0	6.7	6.5	10.4
$6,000-$14,999	700	900	1800	3400	23.3	30.0	39.1	32.1
$15,000 OR MORE	1700	1900	2500	6100	56.7	63.3	54.3	57.5
TOTAL	3000	3000	4600	10600	100.0	100.0	100.0	100.0
TABLE C- 47 DAYTONA BEACH, FLA.								
UNDER $6,000	900	1300	200	2400	40.9	18.8	2.9	15.1
$6,000-$14,999	800	3600	2900	7300	36.4	52.2	42.6	45.9
$15,000 OR MORE	500	2000	3700	6200	22.7	29.0	54.4	39.0
TOTAL	2200	6900	6800	15900	100.0	100.0	100.0	100.0
TABLE C- 48 DECATUR, ILL.								
UNDER $6,000	1500	200	200	1900	26.8	7.4	5.6	16.0
$6,000-$14,999	1600	0	700	2300	28.6	0.0	19.4	19.3
$15,000 OR MORE	2500	2500	2700	7700	44.6	92.6	75.0	64.7
TOTAL	5600	2700	3600	11900	100.0	100.0	100.0	100.0
TABLE C- 49 DUBUQUE, IA AND IOWA CITY, IA								
UNDER $6,000	800	400	0	1200	16.0	10.0	0.0	8.6
$6,000-$14,999	900	700	700	2300	18.0	17.5	14.3	16.5
$15,000 OR MORE	3300	2900	4200	10400	66.0	72.5	85.7	74.8
TOTAL	5000	4000	4900	13900	100.0	100.0	100.0	100.0
TABLE C- 50 EAU CLAIRE, WIS.								
UNDER $6,000	300	400	300	1000	7.3	20.0	6.7	9.4
$6,000-$14,999	1100	1100	1000	3200	26.8	55.0	22.2	30.2
$15,000 OR MORE	2700	500	3200	6400	65.9	25.0	71.1	60.4
TOTAL	4100	2000	4500	10600	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C- 51 EL PASO, TX AND LAS CRUCES, NM

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	0	1000	1800	2800	0.0	14.1	23.7	17.0
$6,000-$14,999	1700	4900	3100	9700	94.4	69.0	40.8	58.8
$15,000 OR MORE	100	1200	2700	4000	5.6	16.9	35.5	24.2
TOTAL	1800	7100	7600	16500	100.0	100.0	100.0	100.0

TABLE C- 52 ELKHART, IND.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1200	200	200	1600	21.8	4.8	5.4	11.9
$6,000-$14,999	1400	700	800	2900	25.5	16.7	21.6	21.6
$15,000 OR MORE	2900	3300	2700	8900	52.7	78.6	73.0	66.4
TOTAL	5500	4200	3700	13400	100.0	100.0	100.0	100.0

TABLE C- 53 ELMIRA, N.Y.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1300	600	100	2000	24.5	15.0	2.7	15.4
$6,000-$14,999	1500	1500	1600	4600	28.3	37.5	43.2	35.4
$15,000 OR MORE	2500	1900	2000	6400	47.2	47.5	54.1	49.2
TOTAL	5300	4000	3700	13000	100.0	100.0	100.0	100.0

TABLE C- 54 FALL RIVER, MASS.-R.I.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1300	400	0	1700	16.2	12.5	0.0	12.5
$6,000-$14,999	2600	900	400	3900	32.5	28.1	16.7	28.7
$15,000 OR MORE	4100	1900	2000	8000	51.2	59.4	83.3	58.8
TOTAL	8000	3200	2400	13600	100.0	100.0	100.0	100.0

TABLE C- 55 FARGO-MOORHEAD, N. DAK.-MINN.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	300	200	800	8.1	9.4	3.3	6.2
$6,000-$14,999	1300	800	1400	3500	35.1	25.0	23.0	26.9
$15,000 OR MORE	2100	2100	4500	8700	56.8	65.6	73.8	66.9
TOTAL	3700	3200	6100	13000	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 56 FAYETTEVILLE, N.C.								
UNDER $6,000	600	2400	2200	5200	25.0	16.7	19.1	18.4
$6,000-$14,999	800	8800	4100	13700	33.3	61.1	35.7	48.4
$15,000 OR MORE	1000	3200	5200	9400	41.7	22.2	45.2	33.2
TOTAL	2400	14400	11500	28300	100.0	100.0	100.0	100.0
TABLE C- 57 FAYETTEVILLE-SPRINGDALE, ARK.								
UNDER $6,000	0	800	0	800	0.0	12.9	0.0	5.2
$6,000-$14,999	1000	2400	2500	5900	47.6	38.7	34.7	38.1
$15,000 OR MORE	1100	3000	4700	8800	52.4	48.4	65.3	56.8
TOTAL	2100	6200	7200	15500	100.0	100.0	100.0	100.0
TABLE C- 58 FITCHBURG-LEOMINSTER, MA AND PITTSFIELD, MA								
UNDER $6,000	400	500	200	1100	6.3	15.2	6.3	8.5
$6,000-$14,999	2300	100	700	3100	35.9	3.0	21.9	24.0
$15,000 OR MORE	3700	2700	2300	8700	57.8	81.8	71.9	67.4
TOTAL	6400	3300	3200	12900	100.0	100.0	100.0	100.0
TABLE C- 59 FLORENCE, ALA.								
UNDER $6,000	400	700	600	1700	20.0	16.3	10.9	14.4
$6,000-$14,999	300	1900	1500	3700	15.0	44.2	27.3	31.4
$15,000 OR MORE	1300	1700	3400	6400	65.0	39.5	61.8	54.2
TOTAL	2000	4300	5500	11800	100.0	100.0	100.0	100.0
TABLE C- 60 FLORENCE, S.C.								
UNDER $6,000	300	300	1000	1600	30.0	7.1	19.6	15.5
$6,000-$14,999	500	1500	1100	3100	50.0	35.7	21.6	30.1
$15,000 OR MORE	200	2400	3000	5600	20.0	57.1	58.8	54.4
TOTAL	1000	4200	5100	10300	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C- 61 FORT COLLINS, COLO.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	200	100	400	700	15.4	2.5	4.8	5.1
$6,000-$14,999	400	1400	1500	3300	30.8	35.0	17.9	24.1
$15,000 OR MORE	700	2500	6500	9700	53.8	62.5	77.4	70.8
TOTAL	1300	4000	8400	13700	100.0	100.0	100.0	100.0

TABLE C- 62 FORT MYERS-CAPE CORAL, FLA.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	0	700	1000	1700	0.0	13.2	12.5	12.1
$6,000-$14,999	400	2000	3300	5700	50.0	37.7	41.2	40.4
$15,000 OR MORE	400	2600	3700	6700	50.0	49.1	46.2	47.5
TOTAL	800	5300	8000	14100	100.0	100.0	100.0	100.0

TABLE C- 63 FORT SMITH, ARK.-OKLA.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1700	700	500	2900	39.5	14.9	5.6	16.1
$6,000-$14,999	1200	1200	3400	5800	27.9	25.5	37.8	32.2
$15,000 OR MORE	1400	2800	5100	9300	32.6	59.6	56.7	51.7
TOTAL	4300	4700	9000	18000	100.0	100.0	100.0	100.0

TABLE C- 64 FORT WALTON BEACH, FL AND PANAMA CITY, FL

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	200	1600	900	2700	10.0	17.6	9.3	13.0
$6,000-$14,999	700	4600	3800	9100	35.0	50.5	39.2	43.8
$15,000 OR MORE	1100	2900	5000	9000	55.0	31.9	51.5	43.3
TOTAL	2000	9100	9700	20800	100.0	100.0	100.0	100.0

TABLE C- 65 GADSDEN, ALA.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	500	600	200	1300	18.5	20.7	5.0	13.5
$6,000-$14,999	1400	1300	1300	4000	51.9	44.8	32.5	41.7
$15,000 OR MORE	800	1000	2500	4300	29.6	34.5	62.5	44.8
TOTAL	2700	2900	4000	9600	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 66 GAINESVILLE, FLA.								
UNDER $6,000	400	700	700	1800	30.8	24.1	9.3	15.4
$6,000-$14,999	100	700	2200	3000	7.7	24.1	29.3	25.6
$15,000 OR MORE	800	1500	4600	6900	61.5	51.7	61.3	59.0
TOTAL	1300	2900	7500	11700	100.0	100.0	100.0	100.0
TABLE C- 67 GALVESTON-TEXAS CITY, TEX.								
UNDER $6,000	1700	1200	300	3200	34.7	17.9	5.5	18.7
$6,000-$14,999	1200	1500	1000	3700	24.5	22.4	18.2	21.6
$15,000 OR MORE	2000	4000	4200	10200	40.8	59.7	76.4	59.6
TOTAL	4900	6700	5500	17100	100.0	100.0	100.0	100.0
TABLE C- 68 GLENS FALLS, N.Y.								
UNDER $6,000	800	200	400	1400	14.5	9.5	22.2	14.9
$6,000-$14,999	1900	800	700	3400	34.5	38.1	38.9	36.2
$15,000 OR MORE	2800	1100	700	4600	50.9	52.4	38.9	48.9
TOTAL	5500	2100	1800	9400	100.0	100.0	100.0	100.0
TABLE C- 69 GREELEY, COLO.								
UNDER $6,000	0	100	200	300	0.0	4.2	2.8	2.4
$6,000-$14,999	1500	500	2000	4000	50.0	20.8	27.8	31.7
$15,000 OR MORE	1500	1800	5000	8300	50.0	75.0	69.4	65.9
TOTAL	3000	2400	7200	12600	100.0	100.0	100.0	100.0
TABLE C- 70 GREEN BAY, WIS.								
UNDER $6,000	500	500	100	1100	10.0	11.6	1.4	6.7
$6,000-$14,999	1600	1100	1300	4000	32.0	25.6	18.3	24.4
$15,000 OR MORE	2900	2700	5700	11300	58.0	62.8	80.3	68.9
TOTAL	5000	4300	7100	16400	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 71 GREENSBORO-WINSTON-SALEM-HIGH POINT, N.C.								
UNDER $6,000	500	800	500	1800	33.3	15.7	8.3	14.3
$6,000-$14,999	500	1500	1200	3200	33.3	29.4	20.0	25.4
$15,000 OR MORE	500	2800	4300	7600	33.3	54.9	71.7	60.3
TOTAL	1500	5100	6000	12600	100.0	100.0	100.0	100.0
TABLE C- 72 HAGERSTOWN, MD.								
UNDER $6,000	400	100	100	600	14.3	4.2	4.5	8.1
$6,000-$14,999	600	1200	500	2300	21.4	50.0	22.7	31.1
$15,000 OR MORE	1800	1100	1600	4500	64.3	45.8	72.7	60.8
TOTAL	2800	2400	2200	7400	100.0	100.0	100.0	100.0
TABLE C- 73 HAMILTON-MIDDLETOWN, OHIO								
UNDER $6,000	1100	400	600	2100	15.1	6.1	6.3	8.9
$6,000-$14,999	4000	1800	1300	7100	54.8	27.3	13.5	30.2
$15,000 OR MORE	2200	4400	7700	14300	30.1	66.7	80.2	60.9
TOTAL	7300	6600	9600	23500	100.0	100.0	100.0	100.0
TABLE C- 74 HARRISBURG, PA.								
UNDER $6,000	2600	1800	500	4900	17.4	18.9	4.5	13.8
$6,000-$14,999	4600	1600	1900	8100	30.9	16.8	17.0	22.8
$15,000 OR MORE	7700	6100	8800	22600	51.7	64.2	78.6	63.5
TOTAL	14900	9500	11200	35600	100.0	100.0	100.0	100.0
TABLE C- 75 HICKORY, N.C.								
UNDER $6,000	200	200	0	400	10.5	6.5	0.0	3.9
$6,000-$14,999	1000	1100	800	2900	52.6	35.5	15.4	28.4
$15,000 OR MORE	700	1800	4400	6900	36.8	58.1	84.6	67.6
TOTAL	1900	3100	5200	10200	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 76 HUNTINGTON-ASHLAND, W. VA.-KY.-OHIO								
UNDER $6,000	2900	1300	1700	5900	25.2	16.5	18.7	20.7
$6,000-$14,999	3000	2300	2300	7600	26.1	29.1	25.3	26.7
$15,000 OR MORE	5600	4300	5100	15000	48.7	54.4	56.0	52.6
TOTAL	11500	7900	9100	28500	100.0	100.0	100.0	100.0
TABLE C- 77 JACKSON, MICH.								
UNDER $6,000	700	100	100	900	10.9	4.0	2.3	6.8
$6,000-$14,999	1600	500	1200	3300	25.0	20.0	27.9	25.0
$15,000 OR MORE	4100	1900	3000	9000	64.1	76.0	69.8	68.2
TOTAL	6400	2500	4300	13200	100.0	100.0	100.0	100.0
TABLE C- 78 JACKSONVILLE, N.C.								
UNDER $6,000	300	800	400	1500	30.0	14.5	7.5	12.7
$6,000-$14,999	400	3800	3200	7400	40.0	69.1	60.4	62.7
$15,000 OR MORE	300	900	1700	2900	30.0	16.4	32.1	24.6
TOTAL	1000	5500	5300	11800	100.0	100.0	100.0	100.0
TABLE C- 79 JANESVILLE-BELOIT, WIS.								
UNDER $6,000	300	0	200	500	5.3	0.0	5.9	4.0
$6,000-$14,999	1900	300	500	2700	33.3	8.6	14.7	21.4
$15,000 OR MORE	3500	3200	2700	9400	61.4	91.4	79.4	74.6
TOTAL	5700	3500	3400	12600	100.0	100.0	100.0	100.0
TABLE C- 80 JOHNSON CITY-KINGSPORT-BRISTOL, TENN.-VA.								
UNDER $6,000	1900	1100	1900	4900	23.2	9.9	11.7	13.8
$6,000-$14,999	2900	4300	4000	11200	35.4	38.7	24.7	31.5
$15,000 OR MORE	3400	5700	10300	19400	41.5	51.4	63.6	54.6
TOTAL	8200	11100	16200	35500	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 81 JOHNSTOWN, PA.								
UNDER $6,000	1600	800	600	3000	12.0	25.0	7.5	12.2
$6,000-$14,999	3700	1000	2200	6900	27.8	31.3	27.5	28.2
$15,000 OR MORE	8000	1400	5200	14600	60.2	43.8	65.0	59.6
TOTAL	13300	3200	8000	24500	100.0	100.0	100.0	100.0
TABLE C- 82 JOPLIN, MO.								
UNDER $6,000	900	0	200	1100	13.8	0.0	10.0	10.1
$6,000-$14,999	2900	1200	700	4800	44.6	50.0	35.0	44.0
$15,000 OR MORE	2700	1200	1100	5000	41.5	50.0	55.0	45.9
TOTAL	6500	2400	2000	10900	100.0	100.0	100.0	100.0
TABLE C- 83 KANKAKEE, ILL.								
UNDER $6,000	800	700	300	1800	22.2	18.9	9.1	17.0
$6,000-$14,999	1200	200	1000	2400	33.3	5.4	30.3	22.6
$15,000 OR MORE	1600	2800	2000	6400	44.4	75.7	60.6	60.4
TOTAL	3600	3700	3300	10600	100.0	100.0	100.0	100.0
TABLE C- 84 KENOSHA, WIS.								
UNDER $6,000	300	0	0	300	5.4	0.0	0.0	2.9
$6,000-$14,999	1100	100	100	1300	19.6	3.4	5.3	12.5
$15,000 OR MORE	4200	2800	1800	8800	75.0	96.6	94.7	84.6
TOTAL	5600	2900	1900	10400	100.0	100.0	100.0	100.0
TABLE C- 85 KILLEEN-TEMPLE, TEX.								
UNDER $6,000	300	700	1700	2700	12.0	8.5	13.2	11.4
$6,000-$14,999	1200	4200	5900	11300	48.0	51.2	45.7	47.9
$15,000 OR MORE	1000	3300	5300	9600	40.0	40.2	41.1	40.7
TOTAL	2500	8200	12900	23600	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN					PERCENT		
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C- 86 KOKOMO, IND.								
UNDER $6,000	700	300	300	1300	18.4	7.7	11.1	12.5
$6,000-$14,999	900	1000	200	2100	23.7	25.6	7.4	20.2
$15,000 OR MORE	2200	2600	2200	7000	57.9	66.7	81.5	67.3
TOTAL	3800	3900	2700	10400	100.0	100.0	100.0	100.0
TABLE C- 87 LA CROSSE, WI AND ROCHESTER, MN								
UNDER $6,000	300	0	500	800	7.7	0.0	7.2	5.2
$6,000-$14,999	800	1500	1000	3300	20.5	32.6	14.5	21.4
$15,000 OR MORE	2800	3100	5400	11300	71.8	67.4	78.3	73.4
TOTAL	3900	4600	6900	15400	100.0	100.0	100.0	100.0
TABLE C- 88 LAFAYETTE, LA.								
UNDER $6,000	400	1100	700	2200	17.4	22.0	7.7	13.4
$6,000-$14,999	300	900	1900	3100	13.0	18.0	20.9	18.9
$15,000 OR MORE	1600	3000	6500	11100	69.6	60.0	71.4	67.7
TOTAL	2300	5000	9100	16400	100.0	100.0	100.0	100.0
TABLE C- 89 LAFAYETTE-WEST LAFAYETTE, IND.								
UNDER $6,000	200	0	400	600	9.1	0.0	11.1	6.7
$6,000-$14,999	800	1200	800	2800	36.4	37.5	22.2	31.1
$15,000 OR MORE	1200	2000	2400	5600	54.5	62.5	66.7	62.2
TOTAL	2200	3200	3600	9000	100.0	100.0	100.0	100.0
TABLE C- 90 LAKE CHARLES, LA.								
UNDER $6,000	600	1700	400	2700	14.6	24.3	6.9	16.0
$6,000-$14,999	1700	1900	1400	5000	41.5	27.1	24.1	29.6
$15,000 OR MORE	1800	3400	4000	9200	43.9	48.6	69.0	54.4
TOTAL	4100	7000	5800	16900	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C- 91 LAKELAND-WINTER HAVEN, FLA.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	600	1100	1700	3400	17.6	11.5	17.5	15.0
$6,000-$14,999	1600	3300	3100	8000	47.1	34.4	32.0	35.2
$15,000 OR MORE	1200	5200	4900	11300	35.3	54.2	50.5	49.8
TOTAL	3400	9600	9700	22700	100.0	100.0	100.0	100.0

TABLE C- 92 LANCASTER, PA.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	2800	800	300	3900	15.2	10.4	3.8	11.5
$6,000-$14,999	5400	1600	1400	8400	29.3	20.8	17.9	24.8
$15,000 OR MORE	10200	5300	6100	21600	55.4	68.8	78.2	63.7
TOTAL	18400	7700	7800	33900	100.0	100.0	100.0	100.0

TABLE C- 93 LAREDO, TX AND VICTORIA, TX

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	800	2800	1500	5100	20.0	31.8	17.2	23.7
$6,000-$14,999	2200	3400	3300	8900	55.0	38.6	37.9	41.4
$15,000 OR MORE	1000	2600	3900	7500	25.0	29.5	44.8	34.9
TOTAL	4000	8800	8700	21500	100.0	100.0	100.0	100.0

TABLE C- 94 LAWTON, OK AND ENID, OK

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	600	900	100	1600	13.0	11.7	1.7	8.7
$6,000-$14,999	3200	3400	2200	8800	69.6	44.2	36.7	48.1
$15,000 OR MORE	800	3400	3700	7900	17.4	44.2	61.7	43.2
TOTAL	4600	7700	6000	18300	100.0	100.0	100.0	100.0

TABLE C- 95 LIMA, OHIO

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	400	500	100	1000	4.2	9.1	2.1	5.1
$6,000-$14,999	3100	1400	1000	5500	32.6	25.5	21.3	27.9
$15,000 OR MORE	6000	3600	3600	13200	63.2	65.5	76.6	67.0
TOTAL	9500	5500	4700	19700	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C- 96 LINCOLN, NEBR.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	500	300	400	1200	10.4	6.8	5.3	7.1
$6,000-$14,999	1900	800	2500	5200	39.6	18.2	32.9	31.0
$15,000 OR MORE	2400	3300	4700	10400	50.0	75.0	61.8	61.9
TOTAL	4800	4400	7600	16800	100.0	100.0	100.0	100.0

TABLE C- 97 LITTLE ROCK, AR AND PINE BLUFF, AR

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	500	800	700	2000	13.5	13.8	13.5	13.6
$6,000-$14,999	1700	1900	1100	4700	45.9	32.8	21.2	32.0
$15,000 OR MORE	1500	3100	3400	8000	40.5	53.4	65.4	54.4
TOTAL	3700	5800	5200	14700	100.0	100.0	100.0	100.0

TABLE C- 98 LONG BRANCH-ASBURY PARK, N.J.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	900	1600	300	2800	9.5	8.9	2.8	7.4
$6,000-$14,999	1800	2400	800	5000	18.9	13.4	7.5	13.2
$15,000 OR MORE	6800	13900	9500	30200	71.6	77.7	89.6	79.5
TOTAL	9500	17900	10600	38000	100.0	100.0	100.0	100.0

TABLE C- 99 LONGVIEW-MARSHALL, TEX.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	400	1900	700	3000	16.7	26.8	12.1	19.6
$6,000-$14,999	1000	1700	900	3600	41.7	23.9	15.5	23.5
$15,000 OR MORE	1000	3500	4200	8700	41.7	49.3	72.4	56.9
TOTAL	2400	7100	5800	15300	100.0	100.0	100.0	100.0

TABLE C-100 LOWELL, MASS.-N.H.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1800	500	0	2300	26.1	8.9	0.0	14.4
$6,000-$14,999	900	500	700	2100	13.0	8.9	20.0	13.1
$15,000 OR MORE	4200	4600	2800	11600	60.9	82.1	80.0	72.5
TOTAL	6900	5600	3500	16000	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-101 LUBBOCK, TEX.								
UNDER $6,000	700	1700	500	2900	24.1	14.7	8.2	14.1
$6,000-$14,999	1300	4400	1100	6800	44.8	37.9	18.0	33.0
$15,000 OR MORE	900	5500	4500	10900	31.0	47.4	73.8	52.9
TOTAL	2900	11600	6100	20600	100.0	100.0	100.0	100.0
TABLE C-102 LYNCHBURG, VA.								
UNDER $6,000	200	700	400	1300	9.1	21.2	7.8	12.3
$6,000-$14,999	500	1300	1300	3100	22.7	39.4	25.5	29.2
$15,000 OR MORE	1500	1300	3400	6200	68.2	39.4	66.7	58.5
TOTAL	2200	3300	5100	10600	100.0	100.0	100.0	100.0
TABLE C-103 MANCHESTER, N.H.								
UNDER $6,000	1000	300	300	1600	15.6	12.0	6.7	11.9
$6,000-$14,999	2200	900	600	3700	34.4	36.0	13.3	27.6
$15,000 OR MORE	3200	1300	3600	8100	50.0	52.0	80.0	60.4
TOTAL	6400	2500	4500	13400	100.0	100.0	100.0	100.0
TABLE C-104 MANSFIELD, OHIO								
UNDER $6,000	400	600	300	1300	10.3	14.3	14.3	12.7
$6,000-$14,999	1100	700	600	2400	28.2	16.7	28.6	23.5
$15,000 OR MORE	2400	2900	1200	6500	61.5	69.0	57.1	63.7
TOTAL	3900	4200	2100	10200	100.0	100.0	100.0	100.0
TABLE C-105 MEDFORD, OREG.								
UNDER $6,000	500	800	900	2200	23.8	12.9	20.0	17.2
$6,000-$14,999	800	1900	1100	3800	38.1	30.6	24.4	29.7
$15,000 OR MORE	800	3500	2500	6800	38.1	56.5	55.6	53.1
TOTAL	2100	6200	4500	12800	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C-106 MELBOURNE-TITUSVILLE-COCOA, FLA.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	0	900	500	1400	0.0	10.2	7.0	8.0
$6,000-$14,999	600	2900	2200	5700	37.5	33.0	31.0	32.6
$15,000 OR MORE	1000	5000	4400	10400	62.5	56.8	62.0	59.4
TOTAL	1600	8800	7100	17500	100.0	100.0	100.0	100.0

TABLE C-107 MIDLAND, TX AND SAN ANGELO, TX

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	900	300	1500	10.3	9.6	8.3	9.4
$6,000-$14,999	1100	2800	700	4600	37.9	29.8	19.4	28.9
$15,000 OR MORE	1500	5700	2600	9800	51.7	60.6	72.2	61.6
TOTAL	2900	9400	3600	15900	100.0	100.0	100.0	100.0

TABLE C-108 MONROE, LA.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	1000	1100	2400	15.8	19.2	17.5	17.9
$6,000-$14,999	1200	1900	1700	4800	63.2	36.5	27.0	35.8
$15,000 OR MORE	400	2300	3500	6200	21.1	44.2	55.6	46.3
TOTAL	1900	5200	6300	13400	100.0	100.0	100.0	100.0

TABLE C-109 MONTGOMERY, ALA.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1600	1900	900	4400	34.0	20.7	8.9	18.3
$6,000-$14,999	2100	4400	2200	8700	44.7	47.8	21.8	36.2
$15,000 OR MORE	1000	2900	7000	10900	21.3	31.5	69.3	45.4
TOTAL	4700	9200	10100	24000	100.0	100.0	100.0	100.0

TABLE C-110 MUNCIE, IND.

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	400	700	200	1300	9.5	14.9	9.5	11.8
$6,000-$14,999	1900	1300	500	3700	45.2	27.7	23.8	33.6
$15,000 OR MORE	1900	2700	1400	6000	45.2	57.4	66.7	54.5
TOTAL	4200	4700	2100	11000	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-111 MUSKEGON-NORTON SHORES-MUSKEGON HEIGHTS, MICH.								
UNDER $6,000	700	500	0	1200	10.9	10.4	0.0	7.6
$6,000-$14,999	2600	1200	900	4700	40.6	25.0	19.6	29.7
$15,000 OR MORE	3100	3100	3700	9900	48.4	64.6	80.4	62.7
TOTAL	6400	4800	4600	15800	100.0	100.0	100.0	100.0
TABLE C-112 NASHUA, N.H.								
UNDER $6,000	0	0	500	500	0.0	0.0	10.9	5.3
$6,000-$14,999	700	300	400	1400	29.2	12.5	8.7	14.9
$15,000 OR MORE	1700	2100	3700	7500	70.8	87.5	80.4	79.8
TOTAL	2400	2400	4600	9400	100.0	100.0	100.0	100.0
TABLE C-113 NEW BEDFORD, MASS.								
UNDER $6,000	1700	0	500	2200	22.1	0.0	13.5	16.4
$6,000-$14,999	2200	500	500	3200	28.6	25.0	13.5	23.9
$15,000 OR MORE	3800	1500	2700	8000	49.4	75.0	73.0	59.7
TOTAL	7700	2000	3700	13400	100.0	100.0	100.0	100.0
TABLE C-114 NEW BRITAIN, CONN.								
UNDER $6,000	400	100	0	500	11.1	3.4	0.0	5.9
$6,000-$14,999	800	900	300	2000	22.2	31.0	15.0	23.5
$15,000 OR MORE	2400	1900	1700	6000	66.7	65.5	85.0	70.6
TOTAL	3600	2900	2000	8500	100.0	100.0	100.0	100.0
TABLE C-115 NEW LONDON-NORWICH, CONN.-R.I.								
UNDER $6,000	1400	500	1200	3100	18.7	6.6	19.0	14.5
$6,000-$14,999	2400	2000	2200	6600	32.0	26.3	34.9	30.8
$15,000 OR MORE	3700	5100	2900	11700	49.3	67.1	46.0	54.7
TOTAL	7500	7600	6300	21400	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-116 NEWARK, OHIO								
UNDER $6,000	600	100	800	1500	13.3	4.3	23.5	14.7
$6,000-$14,999	1600	1100	600	3300	35.6	47.8	17.6	32.4
$15,000 OR MORE	2300	1100	2000	5400	51.1	47.8	58.8	52.9
TOTAL	4500	2300	3400	10200	100.0	100.0	100.0	100.0
TABLE C-117 NEWBURGH-MIDDLETOWN, N.Y.								
UNDER $6,000	700	200	200	1100	6.9	3.8	3.1	5.0
$6,000-$14,999	2900	500	1200	4600	28.4	9.6	18.5	21.0
$15,000 OR MORE	6600	4500	5100	16200	64.7	86.5	78.5	74.0
TOTAL	10200	5200	6500	21900	100.0	100.0	100.0	100.0
TABLE C-118 NEWPORT NEWS-HAMPTON, VA.								
UNDER $6,000	1100	2500	1100	4700	26.2	14.9	11.3	15.3
$6,000-$14,999	1500	5700	3900	11100	35.7	33.9	40.2	36.2
$15,000 OR MORE	1600	8600	4700	14900	38.1	51.2	48.5	48.5
TOTAL	4200	16800	9700	30700	100.0	100.0	100.0	100.0
TABLE C-119 NORTHEAST PENNSYLVANIA								
UNDER $6,000	2800	1000	2100	5900	11.5	15.4	13.9	12.8
$6,000-$14,999	10600	1400	4200	16200	43.4	21.5	27.8	35.2
$15,000 OR MORE	11000	4100	8800	23900	45.1	63.1	58.3	52.0
TOTAL	24400	6500	15100	46000	100.0	100.0	100.0	100.0
TABLE C-120 NORWALK, CONN.								
UNDER $6,000	500	300	0	800	13.5	12.0	0.0	10.8
$6,000-$14,999	600	200	100	900	16.2	8.0	8.3	12.2
$15,000 OR MORE	2600	2000	1100	5700	70.3	80.0	91.7	77.0
TOTAL	3700	2500	1200	7400	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	NUMBER OF CHILDREN 1950-1969	NUMBER OF CHILDREN 1970-1980	TOTAL	PERCENT PRE-1950	PERCENT 1950-1969	PERCENT 1970-1980	TOTAL
TABLE C-121 OCALA, FLA.								
UNDER $6,000	500	400	800	1700	35.7	18.2	17.0	20.5
$6,000-$14,999	600	900	1500	3000	42.9	40.9	31.9	36.1
$15,000 OR MORE	300	900	2400	3600	21.4	40.9	51.1	43.4
TOTAL	1400	2200	4700	8300	100.0	100.0	100.0	100.0
TABLE C-122 ODESSA, TEX.								
UNDER $6,000	0	700	100	800	0.0	9.9	2.6	6.6
$6,000-$14,999	600	1700	1300	3600	50.0	23.9	34.2	29.8
$15,000 OR MORE	600	4700	2400	7700	50.0	66.2	63.2	63.6
TOTAL	1200	7100	3800	12100	100.0	100.0	100.0	100.0
TABLE C-123 OLYMPIA, WASH.								
UNDER $6,000	100	300	1000	1400	7.1	12.5	14.3	13.0
$6,000-$14,999	700	500	1900	3100	50.0	20.8	27.1	28.7
$15,000 OR MORE	600	1600	4100	6300	42.9	66.7	58.6	58.3
TOTAL	1400	2400	7000	10800	100.0	100.0	100.0	100.0
TABLE C-124 PARKERSBURG-MARIETTA, W. VA.-OHIO								
UNDER $6,000	1100	600	800	2500	22.9	13.0	12.7	15.9
$6,000-$14,999	1300	800	1600	3700	27.1	17.4	25.4	23.6
$15,000 OR MORE	2400	3200	3900	9500	50.0	69.6	61.9	60.5
TOTAL	4800	4600	6300	15700	100.0	100.0	100.0	100.0
TABLE C-125 PASCAGOULA-MOSS POINT, MISS.								
UNDER $6,000	100	600	600	1300	25.0	13.0	7.6	10.1
$6,000-$14,999	100	1600	2400	4100	25.0	34.8	30.4	31.8
$15,000 OR MORE	200	2400	4900	7500	50.0	52.2	62.0	58.1
TOTAL	400	4600	7900	12900	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-126 PENSACOLA, FLA.								
UNDER $6,000	1400	2200	1100	4700	45.2	22.7	9.2	19.0
$6,000-$14,999	900	4400	4000	9300	29.0	45.4	33.3	37.5
$15,000 OR MORE	800	3100	6900	10800	25.8	32.0	57.5	43.5
TOTAL	3100	9700	12000	24800	100.0	100.0	100.0	100.0
TABLE C-127 PETERSBURG-COLONIAL HEIGHTS-HOPEWELL, VA.								
UNDER $6,000	200	700	100	1000	11.8	14.9	2.3	9.3
$6,000-$14,999	700	1500	500	2700	41.2	31.9	11.4	25.0
$15,000 OR MORE	800	2500	3800	7100	47.1	53.2	86.4	65.7
TOTAL	1700	4700	4400	10800	100.0	100.0	100.0	100.0
TABLE C-128 PORTLAND, MAINE								
UNDER $6,000	1100	400	400	1900	16.2	11.1	10.8	13.5
$6,000-$14,999	1900	1000	900	3800	27.9	27.8	24.3	27.0
$15,000 OR MORE	3800	2200	2400	8400	55.9	61.1	64.9	59.6
TOTAL	6800	3600	3700	14100	100.0	100.0	100.0	100.0
TABLE C-129 PORTSMOUTH-DOVER-ROCHESTER, N.H.-MAINE								
UNDER $6,000	900	100	100	1100	15.3	3.8	3.6	9.7
$6,000-$14,999	2200	1000	700	3900	37.3	38.5	25.0	34.5
$15,000 OR MORE	2800	1500	2000	6300	47.5	57.7	71.4	55.8
TOTAL	5900	2600	2800	11300	100.0	100.0	100.0	100.0
TABLE C-130 POUGHKEEPSIE, N.Y.								
UNDER $6,000	1300	600	100	2000	17.1	10.2	1.4	9.7
$6,000-$14,999	1900	1500	1800	5200	25.0	25.4	25.0	25.1
$15,000 OR MORE	4400	3800	5300	13500	57.9	64.4	73.6	65.2
TOTAL	7600	5900	7200	20700	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-131 PROVO-OREM, UTAH								
UNDER $6,000	900	900	1700	3500	17.6	10.0	7.1	9.2
$6,000-$14,999	1600	3400	6700	11700	31.4	37.8	27.9	30.7
$15,000 OR MORE	2600	4700	15600	22900	51.0	52.2	65.0	60.1
TOTAL	5100	9000	24000	38100	100.0	100.0	100.0	100.0
TABLE C-132 PUEBLO, COLO.								
UNDER $6,000	400	700	1300	2400	12.5	23.3	25.0	21.1
$6,000-$14,999	1500	900	300	2700	46.9	30.0	5.8	23.7
$15,000 OR MORE	1300	1400	3600	6300	40.6	46.7	69.2	55.3
TOTAL	3200	3000	5200	11400	100.0	100.0	100.0	100.0
TABLE C-133 RACINE, WIS.								
UNDER $6,000	500	700	0	1200	6.3	15.6	0.0	7.0
$6,000-$14,999	1900	800	600	3300	24.1	17.8	12.5	19.2
$15,000 OR MORE	5500	3000	4200	12700	69.6	66.7	87.5	73.8
TOTAL	7900	4500	4800	17200	100.0	100.0	100.0	100.0
TABLE C-134 READING, PA.								
UNDER $6,000	1200	200	1200	2600	10.0	6.5	15.0	11.3
$6,000-$14,999	2900	400	1000	4300	24.2	12.9	12.5	18.6
$15,000 OR MORE	7900	2500	5800	16200	65.8	80.6	72.5	70.1
TOTAL	12000	3100	8000	23100	100.0	100.0	100.0	100.0
TABLE C-135 REDDING, CALIF.								
UNDER $6,000	300	400	300	1000	23.1	10.8	5.5	9.5
$6,000-$14,999	400	1500	2800	4700	30.8	40.5	50.9	44.8
$15,000 OR MORE	600	1800	2400	4800	46.2	48.6	43.6	45.7
TOTAL	1300	3700	5500	10500	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-136 RENO, NEV.								
UNDER $6,000	100	0	400	500	6.7	0.0	5.3	3.8
$6,000-$14,999	600	1500	1500	3600	40.0	35.7	19.7	27.1
$15,000 OR MORE	800	2700	5700	9200	53.3	64.3	75.0	69.2
TOTAL	1500	4200	7600	13300	100.0	100.0	100.0	100.0
TABLE C-137 RICHLAND-KENNEWICK-PASCO, WASH.								
UNDER $6,000	100	0	0	100	4.8	0.0	0.0	0.6
$6,000-$14,999	1100	1700	2300	5100	52.4	31.5	25.6	30.9
$15,000 OR MORE	900	3700	6700	11300	42.9	68.5	74.4	68.5
TOTAL	2100	5400	9000	16500	100.0	100.0	100.0	100.0
TABLE C-138 ROCK HILL, S.C.								
UNDER $6,000	300	200	200	700	15.8	12.5	4.8	9.1
$6,000-$14,999	600	400	1200	2200	31.6	25.0	28.6	28.6
$15,000 OR MORE	1000	1000	2800	4800	52.6	62.5	66.7	62.3
TOTAL	1900	1600	4200	7700	100.0	100.0	100.0	100.0
TABLE C-139 SAGINAW, MICH.								
UNDER $6,000	1300	1200	800	3300	15.5	16.2	12.9	15.0
$6,000-$14,999	1900	600	900	3400	22.6	8.1	14.5	15.5
$15,000 OR MORE	5200	5600	4500	15300	61.9	75.7	72.6	69.5
TOTAL	8400	7400	6200	22000	100.0	100.0	100.0	100.0
TABLE C-140 ST. CLOUD, MINN.								
UNDER $6,000	500	100	200	800	10.9	3.6	2.1	4.7
$6,000-$14,999	1200	700	2400	4300	26.1	25.0	24.7	25.1
$15,000 OR MORE	2900	2000	7100	12000	63.0	71.4	73.2	70.2
TOTAL	4600	2800	9700	17100	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-141 ST. JOSEPH, MO.								
UNDER $6,000	200	0	100	300	4.8	0.0	9.1	3.9
$6,000-$14,999	1300	400	100	1800	31.0	17.4	9.1	23.7
$15,000 OR MORE	2700	1900	900	5500	64.3	82.6	81.8	72.4
TOTAL	4200	2300	1100	7600	100.0	100.0	100.0	100.0
TABLE C-142 SALEM, OREG.								
UNDER $6,000	800	1700	1300	3800	13.6	25.0	11.1	15.6
$6,000-$14,999	1700	1200	3900	6800	28.8	17.6	33.3	27.9
$15,000 OR MORE	3400	3900	6500	13800	57.6	57.4	55.6	56.6
TOTAL	5900	6800	11700	24400	100.0	100.0	100.0	100.0
TABLE C-143 SALISBURY-CONCORD, N.C.								
UNDER $6,000	1100	100	600	1800	20.0	2.7	10.7	12.2
$6,000-$14,999	1300	1500	1800	4600	23.6	40.5	32.1	31.1
$15,000 OR MORE	3100	2100	3200	8400	56.4	56.8	57.1	56.8
TOTAL	5500	3700	5600	14800	100.0	100.0	100.0	100.0
TABLE C-144 SANTA CRUZ, CALIF.								
UNDER $6,000	300	600	700	1600	6.3	10.9	15.6	10.8
$6,000-$14,999	1900	1400	400	3700	39.6	25.5	8.9	25.0
$15,000 OR MORE	2600	3500	3400	9500	54.2	63.6	75.6	64.2
TOTAL	4800	5500	4500	14800	100.0	100.0	100.0	100.0
TABLE C-145 SANTA ROSA, CALIF.								
UNDER $6,000	900	700	600	2200	15.0	7.1	5.8	8.4
$6,000-$14,999	1600	1900	1500	5000	26.7	19.2	14.4	19.0
$15,000 OR MORE	3500	7300	8300	19100	58.3	73.7	79.8	72.6
TOTAL	6000	9900	10400	26300	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-146 SARASOTA, FLA.								
UNDER $6,000	0	500	300	800	0.0	11.9	6.4	8.6
$6,000-$14,999	200	1100	1700	3000	50.0	26.2	36.2	32.3
$15,000 OR MORE	200	2600	2700	5500	50.0	61.9	57.4	59.1
TOTAL	400	4200	4700	9300	100.0	100.0	100.0	100.0
TABLE C-147 SAVANNAH, GA.								
UNDER $6,000	1800	1600	900	4300	33.3	15.1	11.2	17.9
$6,000-$14,999	2100	3600	2400	8100	38.9	34.0	30.0	33.7
$15,000 OR MORE	1500	5400	4700	11600	27.8	50.9	58.7	48.3
TOTAL	5400	10600	8000	24000	100.0	100.0	100.0	100.0
TABLE C-148 SHARON, PA.								
UNDER $6,000	600	400	100	1100	11.3	14.3	3.2	9.8
$6,000-$14,999	1300	300	700	2300	24.5	10.7	22.6	20.5
$15,000 OR MORE	3400	2100	2300	7800	64.2	75.0	74.2	69.6
TOTAL	5300	2800	3100	11200	100.0	100.0	100.0	100.0
TABLE C-149 SHEBOYGAN, WIS.								
UNDER $6,000	300	0	0	300	5.6	0.0	0.0	3.4
$6,000-$14,999	1400	100	600	2100	25.9	8.3	27.3	23.9
$15,000 OR MORE	3700	1100	1600	6400	68.5	91.7	72.7	72.7
TOTAL	5400	1200	2200	8800	100.0	100.0	100.0	100.0
TABLE C-150 SIOUX CITY, IOWA-NEBR.								
UNDER $6,000	700	400	0	1100	14.3	15.4	0.0	9.8
$6,000-$14,999	800	300	800	1900	16.3	11.5	21.6	17.0
$15,000 OR MORE	3400	1900	2900	8200	69.4	73.1	78.4	73.2
TOTAL	4900	2600	3700	11200	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-151 SIOUX FALLS, S. DAK.								
UNDER $6,000	300	200	400	900	12.0	7.4	9.1	9.4
$6,000-$14,999	1200	300	1000	2500	48.0	11.1	22.7	26.0
$15,000 OR MORE	1000	2200	3000	6200	40.0	81.5	68.2	64.6
TOTAL	2500	2700	4400	9600	100.0	100.0	100.0	100.0
TABLE C-152 SPRINGFIELD, ILL.								
UNDER $6,000	1400	500	200	2100	20.3	12.5	3.8	13.0
$6,000-$14,999	1800	1000	700	3500	26.1	25.0	13.2	21.6
$15,000 OR MORE	3700	2500	4400	10600	53.6	62.5	83.0	65.4
TOTAL	6900	4000	5300	16200	100.0	100.0	100.0	100.0
TABLE C-153 SPRINGFIELD, MO.								
UNDER $6,000	500	400	400	1300	10.9	6.9	5.6	7.4
$6,000-$14,999	2300	2400	1700	6400	50.0	41.4	23.6	36.4
$15,000 OR MORE	1800	3000	5100	9900	39.1	51.7	70.8	56.3
TOTAL	4600	5800	7200	17600	100.0	100.0	100.0	100.0
TABLE C-154 SPRINGFIELD, OHIO								
UNDER $6,000	1800	400	600	2800	25.4	9.1	20.7	19.4
$6,000-$14,999	2100	300	600	3000	29.6	6.8	20.7	20.8
$15,000 OR MORE	3200	3700	1700	8600	45.1	84.1	58.6	59.7
TOTAL	7100	4400	2900	14400	100.0	100.0	100.0	100.0
TABLE C-155 STAMFORD, CONN.								
UNDER $6,000	600	400	0	1000	8.5	7.1	0.0	6.6
$6,000-$14,999	1300	900	600	2800	18.3	16.1	25.0	18.5
$15,000 OR MORE	5200	4300	1800	11300	73.2	76.8	75.0	74.8
TOTAL	7100	5600	2400	15100	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C-156 STATE COLLEGE, PA.

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	200	200	700	10.0	11.8	8.0	9.7
$6,000-$14,999	600	500	1200	2300	20.0	29.4	48.0	31.9
$15,000 OR MORE	2100	1000	1100	4200	70.0	58.8	44.0	58.3
TOTAL	3000	1700	2500	7200	100.0	100.0	100.0	100.0

TABLE C-157 STEUBENVILLE-WEIRTON, OHIO-W. VA.

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	500	400	300	1200	8.2	10.5	6.7	8.3
$6,000-$14,999	1100	600	600	2300	18.0	15.8	13.3	16.0
$15,000 OR MORE	4500	2800	3600	10900	73.8	73.7	80.0	75.7
TOTAL	6100	3800	4500	14400	100.0	100.0	100.0	100.0

TABLE C-158 TALLAHASSEE, FLA.

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	100	800	900	1800	6.3	21.1	9.9	12.4
$6,000-$14,999	1000	700	2600	4300	62.5	18.4	28.6	29.7
$15,000 OR MORE	500	2300	5600	8400	31.3	60.5	61.5	57.9
TOTAL	1600	3800	9100	14500	100.0	100.0	100.0	100.0

TABLE C-159 TERRE HAUTE, IND.

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	800	600	500	1900	8.5	17.6	12.5	11.3
$6,000-$14,999	3100	1200	900	5200	33.0	35.3	22.5	31.0
$15,000 OR MORE	5500	1600	2600	9700	58.5	47.1	65.0	57.7
TOTAL	9400	3400	4000	16800	100.0	100.0	100.0	100.0

TABLE C-160 TEXARKANA, TEX.-TEXARKANA, ARK.

FAMILY INCOME	NUMBER OF CHILDREN				PERCENT			
	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	200	300	400	900	8.3	6.5	10.0	8.2
$6,000-$14,999	1100	2000	1000	4100	45.8	43.5	25.0	37.3
$15,000 OR MORE	1100	2300	2600	6000	45.8	50.0	65.0	54.5
TOTAL	2400	4600	4000	11000	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-161 TOLEDO, OHIO-MICH.								
UNDER $6,000	6100	2200	1900	10200	19.0	10.0	11.0	14.3
$6,000-$14,999	8100	3700	2700	14500	25.2	16.9	15.6	20.3
$15,000 OR MORE	17900	16000	12700	46600	55.8	73.1	73.4	65.4
TOTAL	32100	21900	17300	71300	100.0	100.0	100.0	100.0
TABLE C-162 TOPEKA, KS AND LAWRENCE, KS								
UNDER $6,000	500	900	300	1700	17.9	18.8	4.8	12.3
$6,000-$14,999	900	1400	1400	3700	32.1	29.2	22.6	26.8
$15,000 OR MORE	1400	2500	4500	8400	50.0	52.1	72.6	60.9
TOTAL	2800	4800	6200	13800	100.0	100.0	100.0	100.0
TABLE C-163 TRENTON, N.J.								
UNDER $6,000	2500	1100	100	3700	21.9	14.7	3.7	17.1
$6,000-$14,999	3300	1200	0	4500	28.9	16.0	0.0	20.8
$15,000 OR MORE	5600	5200	2600	13400	49.1	69.3	96.3	62.0
TOTAL	11400	7500	2700	21600	100.0	100.0	100.0	100.0
TABLE C-164 TUSCALOOSA, ALA.								
UNDER $6,000	600	1000	400	2000	40.0	21.7	5.8	15.4
$6,000-$14,999	600	900	2500	4000	40.0	19.6	36.2	30.8
$15,000 OR MORE	300	2700	4000	7000	20.0	58.7	58.0	53.8
TOTAL	1500	4600	6900	13000	100.0	100.0	100.0	100.0
TABLE C-165 TYLER, TEX.								
UNDER $6,000	400	200	400	1000	15.4	4.1	8.3	8.1
$6,000-$14,999	1200	1100	1200	3500	46.2	22.4	25.0	28.5
$15,000 OR MORE	1000	3600	3200	7800	38.5	73.5	66.7	63.4
TOTAL	2600	4900	4800	12300	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-166 VINELAND-MILLVILLE-BRIDGETON, N.J.								
UNDER $6,000	1200	500	200	1900	27.3	11.6	7.4	16.7
$6,000-$14,999	1700	1800	300	3800	38.6	41.9	11.1	33.3
$15,000 OR MORE	1500	2000	2200	5700	34.1	46.5	81.5	50.0
TOTAL	4400	4300	2700	11400	100.0	100.0	100.0	100.0
TABLE C-167 VISALIA-TULARE-PORTERVILLE, CALIF.								
UNDER $6,000	1700	1500	900	4100	28.3	14.0	7.1	13.9
$6,000-$14,999	2300	4500	4500	11300	38.3	42.1	35.4	38.4
$15,000 OR MORE	2000	4700	7300	14000	33.3	43.9	57.5	47.6
TOTAL	6000	10700	12700	29400	100.0	100.0	100.0	100.0
TABLE C-168 WACO, TEX.								
UNDER $6,000	400	600	500	1500	8.7	8.8	12.8	9.8
$6,000-$14,999	2200	3400	1100	6700	47.8	50.0	28.2	43.8
$15,000 OR MORE	2000	2800	2300	7100	43.5	41.2	59.0	46.4
TOTAL	4600	6800	3900	15300	100.0	100.0	100.0	100.0
TABLE C-169 WATERLOO-CEDAR FALLS, IOWA								
UNDER $6,000	600	400	400	1400	12.8	10.3	8.2	10.4
$6,000-$14,999	500	400	1200	2100	10.6	10.3	24.5	15.6
$15,000 OR MORE	3600	3100	3300	10000	76.6	79.5	67.3	74.1
TOTAL	4700	3900	4900	13500	100.0	100.0	100.0	100.0
TABLE C-170 WAUSAU, WIS.								
UNDER $6,000	400	200	0	600	9.3	10.0	0.0	5.7
$6,000-$14,999	1000	500	1200	2700	23.3	25.0	27.9	25.5
$15,000 OR MORE	2900	1300	3100	7300	67.4	65.0	72.1	68.9
TOTAL	4300	2000	4300	10600	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

	NUMBER OF CHILDREN				PERCENT			
FAMILY INCOME	PRE-1950	1950-1969	1970-1980	TOTAL	PRE-1950	1950-1969	1970-1980	TOTAL
TABLE C-171 WHEELING, W. VA.-OHIO								
UNDER $6,000	1100	300	100	1500	15.3	10.0	2.6	10.6
$6,000-$14,999	2100	600	1000	3700	29.2	20.0	25.6	26.2
$15,000 OR MORE	4000	2100	2800	8900	55.6	70.0	71.8	63.1
TOTAL	7200	3000	3900	14100	100.0	100.0	100.0	100.0
TABLE C-172 WICHITA FALLS, TEX.								
UNDER $6,000	400	700	200	1300	11.8	11.7	11.1	11.6
$6,000-$14,999	1300	2300	400	4000	38.2	38.3	22.2	35.7
$15,000 OR MORE	1700	3000	1200	5900	50.0	50.0	66.7	52.7
TOTAL	3400	6000	1800	11200	100.0	100.0	100.0	100.0
TABLE C-173 WILLIAMSPORT, PA.								
UNDER $6,000	300	0	300	600	5.3	0.0	11.1	6.1
$6,000-$14,999	3300	300	1000	4600	57.9	21.4	37.0	46.9
$15,000 OR MORE	2100	1100	1400	4600	36.8	78.6	51.9	46.9
TOTAL	5700	1400	2700	9800	100.0	100.0	100.0	100.0
TABLE C-174 WILMINGTON, DEL.-N.J.-MD.								
UNDER $6,000	1600	3000	900	5500	13.1	18.4	7.0	13.3
$6,000-$14,999	3400	4200	1700	9300	27.9	25.8	13.3	22.5
$15,000 OR MORE	7200	9100	10200	26500	59.0	55.8	79.7	64.2
TOTAL	12200	16300	12800	41300	100.0	100.0	100.0	100.0
TABLE C-175 WILMINGTON, N.C.								
UNDER $6,000	200	1400	1300	2900	9.1	35.9	19.7	22.8
$6,000-$14,999	1000	600	2000	3600	45.5	15.4	30.3	28.3
$15,000 OR MORE	1000	1900	3300	6200	45.5	48.7	50.0	48.8
TOTAL	2200	3900	6600	12700	100.0	100.0	100.0	100.0

CENSUS COUNT OF CHILDREN OF ALL RACES 6 MONTHS TO 5 YEARS BY FAMILY INCOME, STATUS AND AGE OF HOUSING, 1980

TABLE C-176 YAKIMA, WASH.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1000	400	900	2300	16.1	8.9	15.3	13.9
$6,000-$14,999	2100	1400	2000	5500	33.9	31.1	33.9	33.1
$15,000 OR MORE	3100	2700	3000	8800	50.0	60.0	50.8	53.0
TOTAL	6200	4500	5900	16600	100.0	100.0	100.0	100.0

TABLE C-177 YORK, PA.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	1000	600	500	2100	7.7	9.4	4.2	6.7
$6,000-$14,999	3200	2200	2700	8100	24.6	34.4	22.9	26.0
$15,000 OR MORE	8800	3600	8600	21000	67.7	56.3	72.9	67.3
TOTAL	13000	6400	11800	31200	100.0	100.0	100.0	100.0

TABLE C-178 YUBA CITY, CALIF.

FAMILY INCOME	NUMBER OF CHILDREN PRE-1950	1950-1969	1970-1980	TOTAL	PERCENT PRE-1950	1950-1969	1970-1980	TOTAL
UNDER $6,000	300	600	100	1000	15.0	10.5	3.2	9.3
$6,000-$14,999	900	2300	1700	4900	45.0	40.4	54.8	45.4
$15,000 OR MORE	800	2800	1300	4900	40.0	49.1	41.9	45.4
TOTAL	2000	5700	3100	10800	100.0	100.0	100.0	100.0

APPENDIX D

SMSAs RANKED BY NUMBER OF YOUNG CHILDREN IN PRE-1950 HOUSING AS OF 1980

This table ranks all SMSAs by the number of children in pre-1950 housing for 1980.

The details shown include the child population distribution by "Inside Central City" (Inside C.C.), "Not In Central City" (Outside C.C.), residential unit age status, and the distribution into three categories of age of housing units.

APPENDIX TABLE D. SMSAs RANKED BY NUMBER OF CHILDREN 6 MONTHS - 5 YEARS LIVING IN PRE-1950 HOUSING, AND DISTRIBUTION OF ALL CHILDREN IN SMSA BY AGE OF HOUSING AND URBAN STATUS WHEN AVAILABLE, 1980 CENSUS

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
1	New York, NY-NJ	Total	422,800	200,900	79,800	60.1	28.6	11.3	703,000
		Inside C.C.	362,900	152,100	56,000	63.6	26.6	9.8	571,000
		Outside C.C.	59,900	48,800	23,800	45.2	36.8	18.0	132,500
2	Chicago, IL	Total	271,500	218,500	138,800	43.2	34.7	22.1	628,800
		Inside C.C.	191,100	74,600	12,600	68.7	26.8	4.5	278,300
		Outside C.C.	80,400	143,900	126,200	22.9	41.1	36.0	350,500
3	Los Angeles-Long Beach, CA	Total	225,700	353,700	93,900	33.5	52.5	13.9	673,300
		Inside C.C.	122,700	133,700	29,500	42.9	46.8	10.3	285,900
		Outside C.C.	103,000	220,000	64,400	26.6	56.8	16.6	387,400
4	Philadelphia, PA-NJ	Total	172,500	119,000	80,300	46.4	32.0	21.6	371,800
		Inside C.C.	91,700	33,000	6,800	69.7	25.1	5.2	131,500
		Outside C.C.	80,800	86,000	73,500	33.6	35.8	30.6	240,300
5	Detroit, MI	Total	141,900	144,000	89,900	37.8	38.3	23.9	375,800
		Inside C.C.	78,600	32,900	2,800	68.8	28.8	2.4	114,300
		Outside C.C.	63,300	111,100	87,100	24.2	42.5	33.3	261,500
6	Boston, MA	Total	110,400	40,900	24,800	62.7	23.2	14.1	176,100
		Inside C.C.	26,800	6,800	2,200	74.9	19.0	6.1	35,800
		Outside C.C.	83,600	34,100	22,600	59.6	24.3	16.1	140,300
7	Newark, NJ	Total	80,300	52,100	18,800	53.1	34.5	12.4	151,200
		Inside C.C.	22,300	10,400	1,900	64.5	30.1	5.5	34,600
		Outside C.C.	58,000	41,700	16,900	49.7	35.8	14.5	116,600
8	Cleveland, OH	Total	75,100	53,700	24,200	49.1	35.1	15.8	153,000
		Inside C.C.	39,600	12,300	1,000	74.9	23.3	1.9	52,900
		Outside C.C.	35,500	41,400	23,200	35.5	41.4	23.2	100,100

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
9	San Francisco-Oakland, CA	Total	74,800	101,900	54,800	32.2	44.0	23.7	231,500
		Inside C.C.	37,200	19,900	5,600	59.3	31.7	8.9	62,700
		Outside C.C.	37,600	82,000	49,200	22.3	48.6	29.1	168,800
10	Pittsburgh, PA	Total	71,300	43,600	35,600	47.4	29.0	23.7	150,500
		Inside C.C.	17,600	5,300	1,700	71.5	21.5	6.9	24,600
		Outside C.C.	53,700	38,300	33,900	42.7	30.4	26.9	125,900
11	St. Louis, MO-IL	Total	69,200	75,900	58,500	34.0	37.3	28.7	203,600
		Inside C.C.	29,800	7,100	2,300	76.0	18.1	5.9	39,200
		Outside C.C.	39,400	68,800	56,200	24.0	41.8	34.2	164,400
12	Minneapolis-St. Paul, MI-WI	Total	60,000	58,800	67,800	32.2	31.5	36.3	186,600
		Inside C.C.	37,200	9,300	4,800	72.5	18.1	9.4	51,300
		Outside C.C.	22,800	49,500	63,000	16.9	36.6	46.6	135,300
13	Baltimore, MD	Total	56,700	58,000	51,000	34.2	35.0	30.8	165,700
		Inside C.C.	37,400	20,100	3,600	61.2	32.9	45.3	61,100
		Outside C.C.	19,300	37,900	47,400	18.5	36.2	23.8	104,600
14	Milwaukee, WI	Total	53,100	40,000	29,000	43.5	32.8	23.8	122,100
		Inside C.C.	35,600	21,100	5,800	57.0	33.8	9.3	62,500
		Outside C.C.	17,500	18,900	23,200	29.4	31.7	38.9	59,600
15	Nassau-Suffolk, NY	Total	51,100	97,700	43,700	26.5	50.8	22.8	192,500
16	Washington, DC-MD-VA	Total	48,900	108,000	77,500	20.9	46.1	33.1	234,400
		Inside C.C.	21,100	15,400	4,300	51.7	37.7	10.5	40,800
		Outside C.C.	27,800	92,600	73,200	14.4	47.8	37.8	193,600
17	Buffalo, NY	Total	46,800	28,200	16,300	51.3	30.9	17.9	91,300
		Inside C.C.	21,900	3,600	300	84.8	14.0	1.2	25,800
		Outside C.C.	24,900	24,600	16,000	38.0	37.6	24.4	65,500

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
18	Cincinnati, OH-KY-IN	Total	44,300	45,400	35,300	35.4	36.3	28.2	125,000
		Inside C.C.	19,300	12,200	2,100	57.4	36.3	6.3	33,600
		Outside C.C.	25,000	33,200	33,200	27.4	36.3	36.3	91,400
19	Dallas-Ft. Worth, TX	Total	40,900	119,600	106,900	15.3	44.7	40.0	267,400
		Inside C.C.	26,700	61,300	24,200	23.8	54.6	21.6	112,200
		Outside C.C.	14,200	58,300	82,700	9.1	37.6	53.3	155,200
20	Houston, TX	Total	36,400	111,700	145,300	12.4	38.1	49.5	293,400
		Inside C.C.	26,600	75,800	50,900	17.4	49.4	33.2	153,300
		Outside C.C.	9,800	35,900	94,400	7.0	25.6	67.4	140,100
21	Rochester, NY	Total	35,900	22,900	20,200	45.4	29.0	25.6	79,000
		Inside C.C.	17,300	3,600	1,400	77.6	16.1	6.3	22,300
		Outside C.C.	18,600	19,300	18,800	32.8	34.0	33.2	56,700
22	Jersey City, NJ	Total	34,200	8,500	4,400	72.6	18.0	9.3	47,100
		Inside C.C.	17,500	4,500	1,300	75.1	19.3	5.6	23,300
		Outside C.C.	16,700	4,000	3,100	70.2	16.8	13.0	23,800
23	Albany-Schenectady-Troy, NY	Total	34,000	14,500	14,100	54.3	23.2	22.5	62,600
		Inside C.C.	13,400	2,800	1,000	77.9	16.3	5.8	17,200
		Outside C.C.	20,600	11,700	13,100	45.4	25.8	28.9	45,400
24	Providence-Warwick-Pawtucket, RI-MA	Total	33,300	16,600	12,600	53.3	26.6	20.2	62,500
		Inside C.C.	15,000	5,000	1,600	69.4	23.1	7.4	21,600
		Outside C.C.	18,300	11,600	11,000	44.7	28.4	26.9	40,900
25	Portland, OR	Total	32,900	31,100	45,800	30.0	28.3	41.7	109,800
		Inside C.C.	18,300	7,200	2,500	65.4	25.7	8.9	28,000
		Outside C.C.	14,600	23,900	43,300	17.8	29.2	52.9	81,800
26	Toledo, OH-MI	Total	32,100	21,900	17,300	45.0	30.7	24.3	71,300

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
27	Columbus, OH	Total	31,500	37,900	34,200	30.4	36.6	33.0	103,600
		Inside C.C.	17,200	21,500	15,700	31.6	39.5	28.9	54,400
		Outside C.C.	14,300	16,400	18,500	29.1	33.3	37.6	49,200
28	Kansas City, MO-KA	Total	31,500	46,900	33,900	28.0	41.8	30.2	112,300
		Inside C.C.	14,900	12,900	4,100	46.7	40.4	12.9	31,900
		Outside C.C.	16,600	34,000	29,800	20.6	42.3	37.1	80,400
29	New Orleans, LA	Total	28,600	49,300	40,100	24.2	41.8	34.0	118,000
		Inside C.C.	23,000	24,800	8,900	40.6	43.7	15.7	56,700
		Outside C.C.	5,600	24,500	31,200	9.1	40.0	50.9	61,300
30	Seattle-Everett, WA	Total	28,600	48,300	45,700	23.3	39.4	37.3	122,600
		Inside C.C.	18,200	10,300	2,400	58.9	33.3	7.8	30,900
		Outside C.C.	10,400	38,000	43,300	11.3	41.4	47.2	91,700
31	Indianapolis, IN	Total	27,700	41,900	30,800	27.6	41.7	30.7	100,400
		Inside C.C.	20,400	27,500	13,200	33.4	45.0	21.6	61,100
		Outside C.C.	7,300	14,400	17,600	18.6	36.6	44.8	39,300
32	Riverside-San Bernadino-Ontario, CA	Total	25,900	68,700	62,000	16.5	43.9	39.6	156,600
		Inside C.C.	8,200	18,700	14,100	20.0	45.6	34.4	41,000
		Outside C.C.	17,700	50,000	47,900	15.3	43.3	41.4	115,600
33	Denver-Boulder, CO	Total	25,300	49,700	67,900	17.7	34.8	47.5	142,900
		Inside C.C.	15,600	17,800	7,800	37.9	43.2	18.9	41,200
		Outside C.C.	9,700	31,900	60,100	9.5	31.4	59.1	101,700
34	Syracuse, NY	Total	24,700	14,500	14,800	45.7	26.9	27.4	54,000
		Inside C.C.	10,300	2,900	300	76.3	21.5	2.2	13,500
		Outside C.C.	14,400	11,600	14,500	35.6	28.6	35.8	40,500
35	Northeast Pennsylvania	Total	24,400	6,500	15,100	53.0	14.1	32.8	46,000

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
36	Gary-Hammond-East Chicago, IN	Total	24,400	25,700	15,400	37.3	39.2	23.5	65,500
		Inside C.C.	17,400	13,200	2,600	52.4	39.8	7.8	33,200
		Outside C.C.	7,000	12,500	12,800	21.7	38.7	39.6	32,300
37	San Diego, CA	Total	23,900	64,700	66,200	15.4	41.8	42.8	154,800
		Inside C.C.	14,200	28,900	23,400	21.4	43.5	35.2	66,500
		Outside C.C.	9,700	35,800	42,800	11.0	40.5	48.5	88,300
38	San Antonio, TX	Total	23,000	45,700	42,500	20.7	41.1	38.2	111,200
		Inside C.C.	19,200	39,300	26,900	22.5	46.0	31.5	85,400
		Outside C.C.	3,800	6,400	15,600	14.7	24.8	60.5	25,800
39	Akron, OH	Total	22,900	20,800	13,200	40.2	36.6	23.2	56,900
		Inside C.C.	13,900	6,800	1,900	61.5	30.1	8.4	22,600
		Outside C.C.	9,000	14,000	11,300	26.2	40.8	32.9	34,300
40	Dayton, OH	Total	22,800	29,500	17,100	32.9	42.5	24.6	69,400
		Inside C.C.	11,500	5,300	1,800	61.8	28.5	9.7	18,600
		Outside C.C.	11,300	24,200	15,300	22.2	47.6	30.1	50,800
41	Hartford, CT	Total	21,900	18,400	12,000	41.9	35.2	22.9	52,300
		Inside C.C.	9,900	2,400	1,000	74.4	18.0	7.5	13,300
		Outside C.C.	12,000	16,000	11,000	30.8	41.0	28.2	39,000
42	Atlanta, GA	Total	21,900	71,900	82,600	12.4	40.8	46.8	176,400
		Inside C.C.	10,600	20,000	6,100	28.9	54.5	16.6	36,700
		Outside C.C.	11,300	51,900	76,500	8.1	37.1	54.8	139,700
43	Allentown-Bethlehem-Easton, PA-NJ	Total	21,600	9,200	11,900	50.6	21.5	27.9	42,700
		Inside C.C.	9,600	2,400	1,600	70.6	17.6	11.8	13,600
		Outside C.C.	12,000	6,800	10,300	41.2	23.4	35.4	29,100
44	Grand Rapids, MI	Total	21,000	17,900	17,900	37.0	31.5	31.5	56,800
		Inside C.C.	11,900	3,900	1,700	68.0	22.3	9.7	17,500
		Outside C.C.	9,100	14,000	16,200	23.2	35.6	41.2	39,300

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
45	Paterson-Clifton-Passaic, NJ	Total	20,600	11,000	3,100	59.4	31.7	8.9	34,700
		Inside C.C.	17,000	5,500	900	72.6	23.5	3.8	23,400
		Outside C.C.	3,600	5,500	2,200	31.9	48.7	19.5	11,300
46	Salt Lake City-Ogden, UT	Total	20,600	44,600	75,300	14.7	31.7	53.6	140,500
		Inside C.C.	13,200	7,600	4,900	51.4	29.6	19.1	25,700
		Outside C.C.	7,400	37,000	70,400	6.4	32.2	61.3	114,800
47	Louisville, KY-IN	Total	19,800	32,900	27,400	24.7	41.1	34.2	80,100
		Inside C.C.	12,100	8,500	1,700	54.3	38.1	7.6	22,300
		Outside C.C.	7,700	24,400	25,700	13.3	42.2	44.5	56,800
48	Norfolk-VA Beach-Portsmouth, VA-NC	Total	19,100	30,500	26,300	25.2	40.2	34.7	75,900
		Inside C.C.	16,000	25,200	19,200	26.5	41.7	31.8	60,400
		Outside C.C.	3,100	5,300	7,100	20.0	34.2	45.8	15,500
49	Flint, MI	Total	19,000	20,300	13,300	36.1	38.6	25.3	52,600
		Inside C.C.	9,400	7,900	800	51.9	43.6	4.4	18,100
		Outside C.C.	9,600	12,400	12,500	27.8	35.9	36.2	34,500
50	Birmingham, AL	Total	18,600	28,500	25,800	25.5	39.1	34.4	72,900
		Inside C.C.	8,800	12,800	3,500	35.1	51.0	13.9	25,100
		Outside C.C.	9,800	15,700	22,300	20.5	32.8	46.6	47,800
51	Youngstown-Warren, OH	Total	18,000	14,300	12,600	40.1	31.8	28.1	44,900
		Inside C.C.	8,500	6,200	200	57.0	41.6	1.3	14,900
		Outside C.C.	9,500	8,100	12,400	31.7	27.0	41.3	30,000
52	Springfield-Chicopee-Holyoke, MA-CT	Total	17,500	9,100	9,900	47.9	24.9	27.1	36,500
		Inside C.C.	12,400	3,700	3,100	64.6	19.3	16.1	19,200
		Outside C.C.	5,100	5,400	6,800	29.5	31.2	39.3	17,300

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
53	Lansing-E. Lansing, MI	Total	17,500	12,000	15,000	39.3	27.0	33.7	44,500
		Inside C.C.	7,000	5,100	2,600	47.6	34.7	17.7	14,700
		Outside C.C.	10,500	6,900	12,400	35.2	23.2	41.6	29,800
54	Fort Wayne, IN	Total	16,500	11,300	9,900	43.8	30.0	26.3	37,700
		Inside C.C.	9,200	5,800	1,500	55.8	35.2	9.1	16,500
		Outside C.C.	7,300	5,500	8,400	34.4	25.9	39.6	21,100
55	Davenport-Rock Island-Moline, IA-IL	Total	15,800	13,000	10,800	39.9	32.8	27.3	39,600
		Inside C.C.	10,600	6,900	4,200	48.8	31.8	19.4	21,700
		Outside C.C.	5,200	6,100	6,600	29.1	34.1	36.9	17,900
56	New Haven-W. Haven, CT	Total	15,600	10,700	6,500	47.6	32.6	19.8	32,800
		Inside C.C.	9,900	3,700	1,400	66.0	24.7	9.3	15,000
		Outside C.C.	5,700	7,000	5,100	32.0	39.3	28.7	17,800
57	Bridgeport, CT	Total	14,800	8,900	4,200	53.0	31.9	15.1	27,900
		Inside C.C.	8,600	3,100	100	72.9	26.3	.8	11,800
		Outside C.C.	6,200	5,800	4,100	38.5	36.0	25.5	16,100
58	Canton, OH	Total	14,400	10,900	9,400	41.5	31.4	27.1	34,700
59	Sacramento, CA	Total	14,100	34,600	37,400	16.4	40.2	43.4	86,100
		Inside C.C.	6,700	8,600	6,300	31.0	39.8	29.2	21,600
		Outside C.C.	7,400	26,000	31,100	11.5	40.3	48.2	64,500
60	Memphis, TN-AR-MS	Total	14,100	41,400	33,800	15.8	46.4	37.8	89,300
		Inside C.C.	10,900	33,700	17,200	17.6	54.5	27.8	61,800
		Outside C.C.	3,200	7,700	16,600	11.6	28.0	60.4	27,500
61	Miami, FL	Total	13,800	59,800	42,500	11.9	51.5	36.6	116,100
		Inside C.C.	6,600	11,300	3,700	30.6	52.3	17.1	21,600
		Outside C.C.	7,200	48,500	38,800	7.6	51.3	41.1	94,500

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
62	Wichita, KS	Total	13,700	17,100	10,900	32.9	41.0	26.1	41,700
		Inside C.C.	10,400	13,000	4,300	37.5	46.9	15.5	27,700
		Outside C.C.	3,300	4,100	6,600	23.6	29.3	47.1	14,000
63	Anaheim-Santa Ana-Garden Grove, CA	Total	13,500	85,900	60,200	8.5	53.8	37.7	159,600
		Inside C.C.	7,900	34,400	11,500	14.7	63.9	21.4	53,800
		Outside C.C.	5,600	51,500	48,700	5.3	48.7	46.0	105,800
64	Worcester, MA	Total	13,400	6,000	6,900	51.0	22.8	26.2	26,300
		Inside C.C.	7,000	2,800	1,000	64.8	25.9	9.3	10,800
		Outside C.C.	6,400	3,200	5,900	41.3	20.6	38.1	15,500
65	Tacoma, WA	Total	13,200	14,700	20,100	27.5	30.6	41.9	48,000
		Inside C.C.	7,700	4,800	3,500	48.1	30.0	21.9	16,000
		Outside C.C.	5,500	9,900	16,600	17.2	30.9	51.9	32,000
66	Omaha, NB-IA	Total	13,100	18,500	18,200	26.3	37.1	36.5	49,800
		Inside C.C.	9,500	10,000	2,600	43.0	45.2	11.8	22,100
		Outside C.C.	3,600	8,500	15,600	13.0	30.7	56.3	27,700
67	Oklahoma City, OK	Total	13,100	31,900	31,900	17.0	41.5	41.5	76,900
		Inside C.C.	7,800	17,700	12,100	20.7	47.1	32.2	37,300
		Outside C.C.	5,300	14,200	19,800	13.5	36.1	50.4	39,300
68	Tampa-St. Petersburg, FL	Total	12,800	39,500	46,500	13.0	40.0	47.0	98,800
		Inside C.C.	8,700	17,900	6,300	26.4	54.4	19.1	32,900
		Outside C.C.	4,100	21,600	40,200	6.2	32.8	61.0	65,900
69	Duluth-Superior, MN-WI	Total	12,600	4,300	8,100	50.4	17.2	32.4	25,000
		Inside C.C.	8,400	17,900	6,300	26.4	54.4	19.1	32,900
		Outside C.C.	4,200	21,600	40,200	6.2	32.8	61.0	65,900

(continued on following page)

APPENDIX TABLE D. (continued)

Rank	SMSA/Status*		Children 6 months - 5 years Number Pre-1950	 1950-69	 1970-80	 Percent Pre-1950	 1950-69	 1970-80	Total
70	South Bend, IN	Total	12,600	9,000	5,900	45.8	32.7	21.5	27,500
		Inside C.C.	6,800	4,500	1,200	54.4	36.0	9.6	12,500
		Outside C.C.	5,800	4,500	4,700	38.7	30.0	31.3	15,000
71	Nashville-Davidson, TN	Total	12,600	29,500	30,400	17.4	40.7	41.9	72,500
		Inside C.C.	7,200	17,800	11,000	20.0	49.4	30.6	36,000
		Outside C.C.	5,400	11,700	19,400	14.8	32.1	53.2	36,500
72	Peoria, IL	Total	12,500	14,900	8,600	34.7	41.4	12.9	36,000
		Inside C.C.	5,300	5,000	2,300	42.1	39.7	18.3	12,600
		Outside C.C.	7,200	9,900	6,300	30,8	42.3	26.9	23,400
73	Fresno, CA	Total	12,500	19,900	16,100	25.8	41.0	33.2	48,500
		Inside C.C.	5,100	9,500	7,300	23.3	43.4	33.3	21,900
		Outside C.C.	7,400	10,400	8,800	27.8	39.1	33.1	26,600
74	Wilmington, DE-NJ-MD	Total	12,200	16,300	12,800	29.5	39.5	31.0	41,300
75	Utica-Rome, NY	Total	12,000	8,100	5,600	46.7	31.5	21.8	25,700
		Inside C.C.	5,700	3,000	900	59.4	31.3	9.4	9,600
		Outside C.C.	6,300	5,100	4,700	39.1	31.7	29.2	16,100
76	Phoenix, AZ	Total	12,000	50,000	65,900	9.4	39.1	51.5	127,900
		Inside C.C.	8,900	28,800	29,400	13.3	42.9	43.8	67,100
		Outside C.C.	3,100	21,200	36,500	5.1	34.9	60.0	60,800
77	Lawrence-Haverhill, MA-NH	Total	11,900	6,100	3,900	54.3	27.9	17.8	21,900
		Inside C.C.	6,800	2,300	1,000	67.3	22.8	9.9	10,100
		Outside C.C.	5,100	3,800	2,900	43.2	32.2	24.6	11,800
78	Tulsa, OK	Total	11,900	25,900	28,300	18.0	39.2	42.8	66,100
		Inside C.C.	6,000	16,100	9,100	40.4	27.7	31.9	31,200
		Outside C.C.	5,900	3,800	2,900	43.2	32.2	24.6	11,800

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
79	Huntington-Ashland, WV-KY-OH	Total	11,500	7,900	9,100	40.4	27.7	31.9	28,500
80	Trenton, NJ	Total	11,400	7,500	2,700	52.8	34.7	12.5	21,600
81	Jacksonville, FL	Total	11,000	31,100	28,800	15.5	43.9	40.6	70,900
		Inside C.C.	8,800	25,600	19,300	16.4	47.7	35.9	53,700
		Outside C.C.	2,200	5,500	9,500	12.8	32.0	55.2	17,200
82	Binghamton, NY-PA	Total	10,900	5,500	3,900	53.7	27.1	19.2	20,300
83	San Jose, CA	Total	10,800	55,600	42,100	10.0	51.2	38.8	108,500
		Inside C.C.	6,200	27,700	31,400	9.5	42.4	48.1	65,300
		Outside C.C.	4,600	27,900	10,700	10.6	64.6	24.8	43,200
84	Newburgh-Middletown, NY	Total	10,200	5,200	6,500	46.6	23.7	29.7	21,900
85	Erie, PA	Total	10,000	6,000	6,700	44.1	26.4	29.5	22,700
		Inside C.C.	5,100	2,800	1,300	55.4	30.4	14.1	9,200
		Outside C.C.	4,900	3,200	5,400	36.3	23.7	40.0	13,500
86	Corpus Christi, TX	Total	9,900	15,600	10,200	27.7	43.7	28.6	35,700
87	New Brunswick-Perth Amboy-Sayreville, NJ	Total	9,800	23,000	10,000	22.9	53.7	23.4	42,800
		Inside C.C.	4,100	3,800	300	50.0	46.3	3.7	8,200
		Outside C.C.	5,700	19,200	9,700	16.5	55.5	28.0	34,600
88	Lexington-Fayette, KY	Total	9,700	9,500	10,700	32.4	31.8	35.8	29,900
		Inside C.C.	4,400	7,200	6,000	25.0	40.9	34.1	17,600
		Outside C.C.	5,300	2,300	4,700	43.1	18.7	38.2	12,300
89	Bakersfield, CA	Total	9,700	22,700	15,800	20.1	47.1	32.8	48,200
		Inside C.C.	2,000	5,200	5,800	15.4	40.0	44.6	13,000
		Outside C.C.	7,700	17,500	10,000	21.9	49.7	28.4	35,200

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
90	Lima, OH	Total	9,500	5,500	4,700	48.2	27.9	23.9	19,700
91	Long Branch-Asbury Pk, NJ	Total	9,500	17,900	10,600	25.0	47.1	27.9	38,000
92	Terre Haute, IN	Total	9,400	3,400	4,000	56.0	20.2	23.8	16,800
93	Spokane, WA	Total	9,400	8,800	14,800	28.5	26.7	44.8	33,000
		Inside C.C.	6,800	3,800	4,300	45.6	25.5	28.9	14,900
		Outside C.C.	2,600	5,000	10,500	14.4	27.6	58.0	18,100
94	Beaumont-Port Arthur-Orange, TX	Total	9,200	17,100	10,000	25.3	47.1	27.5	36,300
		Inside C.C.	7,100	9,800	3,600	34.6	47.8	17.6	20,500
		Outside C.C.	2,100	7,300	6,400	13.3	46.2	40.5	15,800
95	Honolulu, HI	Total	9,200	35,200	25,000	13.3	50.7	36.0	69,400
		Inside C.C.	5,000	13,000	7,800	19.4	50.4	30.2	25,800
		Outside C.C.	4,200	22,200	17,200	9.6	50.9	39.4	43,600
96	Charlotte-Gastonia, NC	Total	9,000	24,300	21,100	16.5	44.7	38.8	54,400
		Inside C.C.	4,800	16,000	9,600	15.8	52.6	31.6	30,400
		Outside C.C.	4,200	8,300	11,500	17.5	34.6	47.9	24,000
97	Evansville, IN-KY	Total	8,700	7,500	9,200	34.3	29.5	36.2	25,400
		Inside C.C.	6,300	3,000	1,400	58.9	28.0	13.1	10,700
		Outside C.C.	2,400	4,500	7,800	16.3	30.6	53.1	14,700
98	Richmond, VA	Total	8,700	17,500	20,600	18.6	37.4	44.0	46,800
		Inside C.C.	6,300	7,100	2,600	39.4	44.4	16.2	16,000
		Outside C.C.	2,400	10,400	18,000	7.8	33.8	58.4	30,800
99	Reading, PA	Total	8,600	1,800	3,300	62.8	13.1	24.1	13,700

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
100	Knoxville, TN	Total	8,500	11,000	15,400	24.4	31.5	44.1	34,900
		Inside C.C.	4,400	4,200	3,200	37.3	35.6	27.1	11,800
		Outside C.C.	4,100	6,800	12,200	17.7	29.4	52.8	23,100
101	Saginaw, MI	Total	8,400	7,400	6,200	38.2	33.6	28.2	22,000
102	Kalamazoo-Portage, MI	Total	8,400	7,900	8,400	34.0	32.0	34.0	24,700
		Inside C.C.	3,300	4,000	2,000	35.5	43.0	21.5	9,300
		Outside C.C.	5,100	3,900	6,400	33.1	25.3	41.6	15,400
103	Rockford, IL	Total	8,300	10,700	7,400	31.4	40.5	28.0	26,400
		Inside C.C.	5,600	5,100	1,900	44.4	40.5	15.1	12,600
		Outside C.C.	2,700	5,600	5,500	19.6	40.6	39.9	13,800
104	Waterbury, CT	Total	8,200	5,400	5,500	42.9	28.3	28.8	19,100
		Inside C.C.	5,700	1,700	1,400	64.8	19.3	15.9	8,800
		Outside C.C.	2,500	3,700	4,100	24.3	35.9	39.8	10,300
105	Johnson City-Kingsport-Bristol, TN-VA	Total	8,200	11,100	16,200	23.1	31.3	45.6	35,500
106	Appleton-Oshkosh, WI	Total	8,100	7,700	11,700	29.5	28.0	42.5	27,500
		Inside C.C.	3,700	3,000	3,600	35.9	29.1	35.0	10,300
		Outside C.C.	4,400	4,700	8,100	25.6	27.3	47.1	17,200
107	Fall River, MA-RI	Total	8,000	3,200	2,400	58.8	23.5	17.6	13,600
108	Racine, WI	Total	7,900	4,500	4,800	45.9	26.2	27.9	17,200
109	Lorain-Elyria, OH	Total	7,900	7,800	8,900	32.1	31.7	36.2	24,600
		Inside C.C.	5,700	4,700	4,300	38.8	32.0	29.3	14,700
		Outside C.C.	2,200	3,100	4,600	22.2	31.3	46.5	9,900

(continued on following page)

APPENDIX TABLE D. (continued)

Rank	SMSA/Status*		Children 6 months - 5 years Number Pre-1950	1950-69	1970-80	Percent Pre-1950	1950-69	1970-80	Total
110	Greenville-Spartanburg, SC	Total	7,800	17,500	22,500	16.3	36.6	47.1	47,800
		Inside C.C.	1,700	3,200	2,100	24.3	45.7	30.0	7,000
		Outside C.C.	6,100	14,300	20,400	15.0	35.0	50.0	40,800
111	New Bedford, MA	Total	7,700	2,000	3,700	57.5	14.9	27.6	13,400
112	Battle Creek, MI	Total	7,600	6,300	2,700	45.8	38.0	16.3	16,600
113	Poughkeepsie, NY	Total	7,600	5,900	7,200	36.7	28.5	34.8	20,700
114	Shreveport, LA	Total	7,600	18,500	12,100	19.9	48.4	31.7	38,200
		Inside C.C.	4,500	10,200	5,000	22.8	51.8	25.4	19,700
		Outside C.C.	3,100	8,300	7,100	16.8	44.9	38.4	18,500
115	New London-Norwich, CT-RI	Total	7,500	7,600	6,300	35.0	35.5	29.4	21,400
116	Des Moines, IA	Total	7,500	9,900	9,800	26.6	36.4	36.0	27,200
		Inside C.C.	6,300	5,300	3,100	42.9	36.1	21.1	14,700
		Outside C.C.	1,200	4,600	6,700	9.6	36.8	53.6	12,500
117	Stockton, CA	Total	7,500	13,900	13,600	21.4	39.7	38.9	35,000
		Inside C.C.	2,900	6,600	6,600	18.0	41.0	41.0	16,100
		Outside C.C.	4,600	7,300	7,000	24.3	38.6	37.0	18,900
118	Hamilton-Middletown, OH	Total	7,300	6,600	9,600	31.1	28.1	40.9	23,500
119	Wheeling, WV-OH	Total	7,200	3,000	3,900	51.1	21.3	27.7	14,100
120	Springfield, OH	Total	7,100	4,400	2,900	49.3	30.6	20.1	14,400
121	Stamford, CT	Total	7,100	5,600	2,400	47.0	37.1	15.9	15,100
122	Lowell, MA-NH	Total	6,900	5,600	3,500	43.1	35.0	21.9	16,000
123	Springfield, IL	Total	6,900	4,000	5,300	42.6	24.7	32.7	16,200

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
124	Mobile, AL	Total	6,900	16,400	21,300	15.5	36.8	47.8	44,600
		Inside C.C.	3,900	8,900	5,200	21.7	49.4	28.9	18,000
		Outside C.C.	3,000	7,500	16,100	11.3	28.2	60.5	26,600
125	Portland, ME	Total	6,800	3,600	3,700	48.2	25.5	26.2	14,100
126	Joplin, MO	Total	6,500	2,400	2,000	59.6	22.0	18.3	10,900
127	Jackson, MI	Total	6,400	2,500	4,300	48.5	18.9	32.6	13,200
128	Manchester, NH	Total	6,400	2,500	4,500	47.8	18.7	33.6	13,400
129	Muskegon-Norton Shores-Muskegon Heights, MI	Total	6,400	4,800	4,600	40.5	30.4	29.1	15,800
130	Charleston, WV	Total	6,400	7,100	7,600	30.3	33.6	36.0	21,100
131	Charleston-North Charleston, SC	Total	6,400	12,500	21,700	15.8	30.8	53.4	40,600
		Inside C.C.	3,400	4,000	3,500	31.2	36.7	32.1	10,900
		Outside C.C.	3,000	8,500	18,200	10.1	28.6	61.3	29,700
132	Anderson, IN	Total	6,200	5,100	1,800	47.3	38.9	13.7	13,100
133	Yakima, WA	Total	6,200	4,500	5,900	37.3	27.1	35.5	16,600
134	McAllen-Pharr-Edinburg, TX	Total	6,200	11,800	17,500	17.5	33.2	49.3	35,500
		Inside C.C.	2,300	5,600	6,500	16.0	38.9	45.1	14,400
		Outside C.C.	3,900	6,200	11,000	18.5	29.4	52.1	21,100
135	Austin, TX	Total	6,200	13,700	25,600	13.6	30.1	56.3	45,500
		Inside C.C.	4,100	10,000	14,200	14.5	35.3	50.2	28,300
		Outside C.C.	2,100	3,700	11,400	12.2	21.5	66.3	17,200
136	Steubenville-Weirton, OH-WV	Total	6,100	3,800	4,500	42.4	26.4	31.3	14,400

(continued on following page)

APPENDIX TABLE D. (continued)

Rank	SMSA/Status*		Children 6 months - 5 years						
			Number			Percent			
			Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
137	Chattanooga, TN-GA	Total	6,100	11,300	10,200	22.1	40.9	37.0	27,600
		Inside C.C.	4,300	7,200	2,200	31.4	52.6	16.1	13,700
		Outside C.C.	1,800	4,100	8,000	12.9	29.5	57.6	13,900
138	Santa Rosa, CA	Total	6,000	9,900	10,400	22.8	37.6	39.5	26,300
139	Visalia-Tulare-Porterville, CA	Total	6,000	10,700	12,700	20.4	36.4	43.2	29,400
140	Portsmouth-Dover-Rochester, NH-ME	Total	5,900	2,600	2,800	52.2	23.0	24.8	11,300
141	Salem, OR	Total	5,900	6,800	11,700	24.2	27.9	48.0	24,400
142	Williamsport, PA	Total	5,700	1,400	2,700	58.2	14.3	27.6	9,800
143	Janesville-Beloit, WI	Total	5,700	3,500	3,400	45.2	27.8	27.0	12,600
144	Harrisburg, PA	Total	5,600	2,400	2,200	54.9	23.5	21.6	10,200
145	Kenosha, WI	Total	5,600	2,900	1,900	53.8	27.9	18.3	10,400
146	Decatur, IL	Total	5,600	2,700	3,600	47.1	22.7	30.3	11,900
147	Modesto, CA	Total	5,600	8,600	10,900	22.3	34.3	43.3	25,100
		Inside C.C.	1,400	4,300	4,700	13.5	41.3	45.2	10,400
		Outside C.C.	4,200	4,300	6,200	28.6	29.3	42.2	14,700
148	Glens Falls, NY	Total	5,500	2,100	1,800	58.5	22.3	19.1	9,400
149	Elkhart, IN	Total	5,500	4,200	3,700	41.0	31.3	27.6	13,400
150	Salisbury-Concord, NC	Total	5,500	3,700	5,600	37.2	25.0	37.8	14,800
151	Sheboygan, WI	Total	5,400	1,200	2,200	61.4	13.6	25.0	8,800
152	Johnstown, PA	Total	5,400	1,800	2,300	56.8	18.9	24.2	9,500

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
153	Lancaster, PA	Total	5,400	2,800	1,900	53.5	27.7	18.8	10,100
154	Savannah, GA	Total	5,400	10,600	8,000	22.6	43.9	33.5	24,000
155	Sharon, PA	Total	5,300	2,800	3,100	47.3	25.0	27.7	11,200
156	Elmira, NY	Total	5,300	4,000	3,700	40.8	30.8	28.5	13,000
157	Salinas-Seaside-Monterey, CA	Total	5,300	12,300	10,500	18.9	43.8	37.4	28,100
		Inside C.C.	2,400	6,800	4,700	17.3	48.9	33.8	13,900
		Outside C.C.	2,900	5,500	5,800	20.4	38.7	40.9	14,200
158	Raleigh-Durham, NC	Total	5,300	13,600	20,100	13.6	34.9	51.5	39,000
		Inside C.C.	3,500	7,600	5,400	21.2	46.1	32.7	16,500
		Outside C.C.	1,800	6,000	14,700	8.0	26.7	65.3	22,500
159	Baton Rouge, LA	Total	5,300	19,200	24,600	10.8	39.1	50.1	49,100
		Inside C.C.	2,800	10,500	5,300	15.1	56.5	28.5	18,600
		Outside C.C.	2,500	8,700	19,300	8.2	28.5	63.3	30,500
160	Vallejo-Fairfield-Napa, CA	Total	5,200	11,800	13,900	16.8	38.2	45.0	30,900
		Inside C.C.	4,300	8,100	6,700	22.5	42.4	35.1	19,100
		Outside C.C.	900	3,700	7,200	7.6	31.4	61.0	11,800
161	Benton Harbor, MI	Total	5,100	6,500	3,700	32.9	42.8	24.3	15,300
162	Provo-Orem, UT	Total	5,100	9,000	24,000	13.4	23.6	63.0	38,100
163	Orlando, FL	Total	5,100	22,800	26,700	9.3	41.8	48.9	54,600
		Inside C.C.	1,300	6,800	2,600	12.1	63.6	24.3	10,700
		Outside C.C.	3,800	16,000	24,100	8.7	36.4	54.9	43,900
164	Altoona, PA	Total	5,000	2,100	4,100	44.6	18.8	36.6	11,200
165	Green Bay, WI	Total	5,000	4,300	7,100	30.5	26.2	43.3	16,400

(continued on following page)

APPENDIX TABLE D. (continued)

Rank	SMSA/Status*		Children 6 months - 5 years						
			Number			Percent			
			Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
166	Ann Arbor, MI	Total	5,000	11,700	6,200	21.8	51.1	27.1	22,900
		Inside C.C.	2,200	5,100	2,000	23.7	54.8	21.5	9,300
		Outside C.C.	2,800	6,600	4,200	20.6	48.5	30.9	13,600
167	Jackson, MS	Total	5,000	13,800	11,900	16.3	45.0	38.8	30,700
		Inside C.C.	3,200	10,300	4,600	17.7	56.9	25.4	18,100
		Outside C.C.	1,800	3,500	7,300	14.3	27.8	58.0	12,600
168	Sioux City, IA-NB	Total	4,900	2,600	3,700	43.8	23.2	33.0	11,200
169	Galveston-Texas City, TX	Total	4,900	6,700	5,500	28.7	39.2	32.2	17,100
170	Santa Cruz, CA	Total	4,800	5,500	4,500	32.4	37.2	30.4	14,800
171	Parkersburg-Marietta, WV-OH	Total	4,800	4,600	6,300	30.6	29.3	40.1	15,700
172	Lincoln, NB	Total	4,800	4,400	7,600	28.6	26.2	45.2	16,800
173	Madison, WI	Total	4,800	7,700	10,800	20.6	33.0	46.4	23,300
		Inside C.C.	2,600	4,000	3,700	25.2	38.8	35.9	10,300
		Outside C.C.	2,200	3,700	7,100	16.9	28.5	54.6	13,000
174	Oxnard-Simi Valley-Ventura, CA	Total	4,800	24,200	19,900	9.8	48.5	40.7	48,900
		Inside C.C.	1,400	14,000	9,800	5.6	55.6	38.9	25,200
		Outside C.C.	3,400	10,200	10,100	14.3	43.0	42.6	23,700
175	Waterloo-Cedar Falls, IO	Total	4,700	3,900	4,900	34.8	28.9	36.3	13,500
176	Montgomery, AL	Total	4,700	9,200	10,100	19.6	38.3	42.1	24,000
177	Brownsville-Harlingen-San Benito, TX	Total	4,700	11,000	10,900	17.7	41.4	41.0	26,600
178	Waco, TX	Total	4,600	6,800	3,900	30.1	44.4	25.5	15,300

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
179	St. Cloud, MN	Total	4,600	2,800	9,700	26.9	16.4	56.7	17,100
180	Springfield, MO	Total	4,600	5,800	7,200	26.1	33.0	40.9	17,600
181	Newark, OH	Total	4,500	2,300	3,400	44.1	22.5	33.3	10,200
182	Vineland-Milville-Bridgeton, NJ	Total	4,400	4,300	2,700	38.6	37.7	23.7	11,400
183	Brockton, MA	Total	4,400	4,100	3,600	36.4	33.9	29.8	12,100
184	Cedar Rapids, IO	Total	4,400	5,200	4,800	30.6	36.1	33.3	14,400
185	Santa Barbara-Santa Maria-Lompoc, CA	Total	4,400	12,800	4,900	19.9	57.9	22.2	22,100
		Inside C.C.	2,300	6,200	2,100	21.7	58.5	19.8	10,600
		Outside C.C.	2,100	6,600	2,800	18.3	57.4	24.3	11,500
186	Bloomington-Normal, IL	Total	4,300	1,200	3,900	45.7	12.8	41.5	9,400
187	Wausau, WI	Total	4,300	2,000	4,300	40.6	18.9	40.6	10,600
188	Fort Smith, AR-OK	Total	4,300	4,700	9,000	23.9	26.1	50.0	18,000
189	Cumberland, MD-WV	Total	4,200	2,000	1,100	57.5	27.4	15.1	7,300
190	St. Joseph, MO	Total	4,200	2,300	1,100	55.3	30.3	14.5	7,600
191	Muncie, IN	Total	4,200	4,700	2,100	38.2	42.7	19.1	11,000
192	Albuquerque, NM	Total	4,200	15,700	20,100	10.5	39.3	50.2	40,000
		Inside C.C.	2,700	10,900	14,100	9.7	39.4	50.9	27,700
		Outside C.C.	1,500	4,800	6,000	12.2	39.0	48.8	12,300
193	York, PA	Total	4,100	1,700	1,100	59.4	24.6	15.9	6,900

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
194	Eau Claire, WI	Total	4,100	2,000	4,500	38.7	18.9	42.5	10,600
195	Atlantic City, NJ	Total	4,100	4,100	5,700	29.5	29.5	41.0	13,900
196	Lake Charles, LA	Total	4,100	7,000	5,800	24.3	41.4	34.3	16,000
197	Eugene-Springfield, OR	Total	4,000	6,300	12,400	17.6	27.8	54.6	22,700
		Inside C.C.	1,100	2,500	6,100	11.3	25.8	62.9	9,700
		Outside C.C.	2,900	3,800	6,300	22.3	28.2	48.5	13,000
198	Mansfield, OH	Total	3,900	4,200	2,100	38.2	41.2	20.6	10,200
199	Amarillo, TX	Total	3,900	8,800	5,300	21.7	48.9	29.4	18,000
200	Columbia, SC	Total	3,900	11,900	17,700	11.6	35.5	52.8	33,500
		Inside C.C.	2,600	3,300	500	40.6	51.6	7.8	6,400
		Outside C.C.	1,300	8,600	17,200	4.8	31.7	63.5	27,100
201	Kokomo, IN	Total	3,800	3,900	2,700	36.5	37.5	26.0	10,400
202	Norwalk, CT	Total	3,700	2,500	1,200	50.0	33.8	16.2	7,400
203	Fargo-Moorhead, ND-MN	Total	3,700	3,200	6,100	28.5	24.6	46.9	13,000
204	Alexandria, LA	Total	3,700	7,800	6,100	21.0	44.3	34.7	17,600
205	New Britain, CT	Total	3,600	2,900	2,000	42.4	34.1	23.5	8,500
206	Kankakee, IL	Total	3,600	3,700	3,300	34.0	34.9	31.1	10,600
207	Wichita Falls, TX	Total	3,400	6,000	1,800	30.4	53.6	16.1	11,200

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
208	Roanoke, VA	Total	3,400	7,600	6,800	19.1	42.7	38.2	17,800
		Inside C.C.	2,200	4,200	1,800	26.8	51.2	22.0	8,200
		Outside C.C.	1,200	3,400	5,000	12.5	35.4	52.1	9,600
209	Lakeland-Winter Haven, FL	Total	3,400	9,600	9,700	15.0	42.3	42.7	22,700
210	Augusta, GA-SC	Total	3,300	13,000	13,700	11.0	43.3	45.7	30,000
211	Bellingham, WA	Total	3,200	1,600	3,600	38.1	19.0	42.9	8,400
212	Abilene, TX	Total	3,200	5,300	2,200	29.9	49.5	20.6	10,700
213	Pueblo, CO	Total	3,200	3,000	5,200	28.1	26.3	45.6	11,400
214	Columbus, GA-AL	Total	3,200	12,500	6,300	14.5	56.8	28.6	22,000
215	Macon, GA	Total	3,200	12,100	10,100	12.6	47.6	39.8	25,400
		Inside C.C.	2,600	6,600	2,500	22.2	56.4	21.4	11,700
		Outside C.C.	600	5,500	7,600	4.4	40.1	55.5	13,700
216	Anniston, AL	Total	3,100	3,400	3,700	30.4	33.3	36.3	10,200
217	Biloxi-Gulfport, MS	Total	3,100	8,500	6,500	17.1	47.0	35.9	18,100
218	Pensacola, FL	Total	3,100	9,700	12,000	12.5	39.1	48.4	24,800
219	Huntsville, AL	Total	3,100	14,000	9,400	11.7	52.8	35.5	26,500
		Inside C.C.	600	8,100	2,200	5.5	74.3	20.2	10,900
		Outside C.C.	2,500	5,900	7,200	16.0	37.8	46.2	15,600
220	State College, PA	Total	3,000	1,700	2,500	41.7	23.6	34.7	7,200
221	Danville, VA	Total	3,000	3,00	4,600	28.3	28.3	43.4	10,600
222	Greeley, CO	Total	3,000	2,400	7,200	23.8	19.0	57.1	12,600

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
223	Fort Lauderdale-Hollywood, FL	Total	3,000	28,800	29,500	4.9	47.0	48.1	61,300
		Inside C.C.	2,100	9,400	4,600	13.0	58.4	28.6	16,300
		Outside C.C.	900	19,400	24,900	2.0	42.9	55.1	45,200
224	Danbury, CT	Total	2,900	3,600	4,900	25.4	31.6	43.0	11,400
225	Lubbock, TX	Total	2,900	11,600	6,100	14.1	56.3	29.6	20,600
226	Hagerstown, MD	Total	2,800	2,400	2,200	37.8	32.4	29.7	7,400
227	Anderson, SC	Total	2,800	3,600	4,000	26.9	34.6	38.5	10,400
228	Gadsden, AL	Total	2,700	2,900	4,000	28.1	30.2	41.7	9,600
229	Bay City, MI	Total	2,700	3,400	4,500	25.5	32.1	42.5	10,600
230	Billings, MT	Total	2,700	3,000	5,100	25.0	27.8	47.2	10,800
231	Champaign-Urbana-Rantoul, IL	Total	2,700	6,300	3,900	20.9	48.8	30.2	12,900
232	Colorado Springs, CO	Total	2,700	11,900	14,900	9.2	40.3	50.5	29,500
		Inside C.C.	1,700	6,600	9,500	9.6	37.1	53.4	17,800
		Outside C.C.	1,000	5,300	5,400	8.5	45.3	46.2	11,700
233	Tuscon, AZ	Total	2,700	18,800	21,600	6.3	43.6	50.1	43,100
		Inside C.C.	2,200	14,200	11,300	7.9	51.3	40.8	27,700
		Outside C.C.	500	4,600	10,300	3.2	29.9	66.9	15,400
234	Tyler, TX	Total	2,600	4,900	4,800	21.1	39.8	39.0	12,300
235	Bremerton, WA	Total	2,600	2,300	8,900	18.8	16.7	64.5	13,800
236	Sioux Falls, SD	Total	2,500	2,700	4,400	26.0	28.1	45.8	9,600
237	Boise City, ID	Total	2,500	2,700	11,200	15.2	16.5	68.3	16,400

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
238	Killeen-Temple, TX	Total	2,500	8,200	12,900	10.6	34.7	54.7	23,600
239	Nashua, NH	Total	2,400	2,400	4,600	25.5	25.5	48.9	9,400
240	Texarkana, TX-Texarkana, AR	Total	2,400	4,600	4,000	21.8	41.8	36.4	11,000
241	Asheville, NC	Total	2,400	4,200	5,400	20.0	35.0	45.0	12,000
242	Longview-Marshall, TX	Total	2,400	7,100	5,800	15.7	46.4	37.9	15,300
243	Fayetteville, NC	Total	2,400	14,400	11,500	8.5	50.9	40.6	28,300
244	Lafayette, LA	Total	2,300	5,000	9,100	14.0	30.5	55.5	16,400
245	Lafayette-West Lafayette, IN	Total	2,200	3,200	3,600	24.4	35.6	40.0	9,000
246	Lynchburg, VA	Total	2,200	3,300	5,100	20.8	31.1	48.1	10,600
247	Wilmington, NC	Total	2,200	3,900	6,600	17.3	30.7	52.0	12,700
248	Daytona Beach, FL	Total	2,200	6,900	6,800	13.8	43.3	42.8	15,900
249	West Palm Beach-Boca Raton, FL	Total	2,200	16,200	17,200	6.2	45.5	48.3	35,600
		Inside C.C.	900	3,400	2,100	14.1	53.1	32.8	6,400
		Outside C.C.	1,300	12,800	15,100	4.5	43.8	51.7	29,200
250	Medford, OR	Total	2,100	6,200	4,500	16.4	48.4	35.2	12,800
251	Fayetteville-Springdale, AR	Total	2,100	6,200	7,200	13.5	40.0	46.5	15,500
252	Richland-Kennewick-Pasco, WA	Total	2,100	5,400	9,000	12.7	32.7	54.5	16,500
253	Burlington, VT	Total	2,000	3,100	4,500	20.8	32.3	46.9	9,600
254	Yuba City, CA	Total	2,000	5,700	3,100	18.5	52.8	28.7	10,800

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
255	Florence, AL	Total	2,000	4,300	5,500	16.9	36.4	46.6	11,800
256	Rock Hill, SC	Total	1,900	1,600	4,200	24.7	20.8	54.5	7,700
257	Hickory, NC	Total	1,900	3,100	5,200	18.6	30.4	51.0	10,200
258	Monroe, LA	Total	1,900	5,200	6,300	14.2	38.8	47.0	13,400
259	Chico, CA	Total	1,800	4,400	4,200	17.3	42.3	40.4	10,400
260	Bradenton, FL	Total	1,700	3,000	4,800	17.9	31.6	50.5	9,500
261	Petersburg-Colonial Heights-Hopewell, VA	Total	1,700	4,700	4,400	15.7	43.5	40.7	10,800
262	Athens, GA	Total	1,700	4,100	5,100	15.6	37.6	46.8	10,900
263	Newport News-Hampton, VA	Total	1,700	10,300	5,400	9.8	59.2	31.0	17,400
264	Las Vegas, NV	Total	1,700	15,400	22,900	4.3	38.5	57.3	40,000
		Inside C.C.	200	7,400	6,500	1.4	52.5	46.1	14,100
		Outside C.C.	1,500	8,000	16,400	5.8	30.9	63.3	25,900
265	Tallahassee, FL	Total	1,600	3,800	9,100	11.0	26.2	62.8	14,500
266	Melbourne-Titusville-Cocoa, FL	Total	1,600	8,800	7,100	9.2	50.9	41.0	17,300
267	Albany, GA	Total	1,500	4,800	5,500	12.7	40.7	46.6	11,800
268	Tuscaloosa, AL	Total	1,500	4,600	6,900	11.5	35.4	53.1	13,000
269	Reno, NV	Total	1,500	4,200	7,600	11.3	31.6	57.1	13,300
270	Ocala, FL	Total	1,400	2,200	4,700	16.9	26.5	56.6	8,300

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
271	Olympia, WA	Total	1,400	2,400	7,000	13.0	22.2	64.8	10,800
272	Anchorage, AK	Total	1,400	8,600	10,100	7.0	42.8	50.2	20,100
273	Redding, CA	Total	1,300	3,700	5,500	12.4	35.2	52.4	10,500
274	Gainesville, FL	Total	1,300	2,900	7,500	11.1	24.8	64.1	11,700
275	Fort Collins, CO	Total	1,300	4,000	8,400	9.5	29.2	61.3	13,700
276	Clarksville-Hopkinsville, TN-KY	Total	1,300	5,400	7,600	9.1	37.8	53.1	14,300
277	Odessa, TX	Total	1,200	7,100	3,800	9.9	58.7	31.4	12,100
278	Florence, SC	Total	1,000	4,200	5,100	9.7	40.8	49.5	10,300
279	Jacksonville, NC	Total	1,000	5,500	5,300	8.5	46.6	44.9	11,800
280	Charlottesville, VA	Total	900	2,000	3,600	13.8	30.8	55.4	6,500
281	Fort Myers-Cape Coral, FL	Total	800	5,300	8,000	5.7	37.6	56.7	14,100
282	Columbia, MO	Total	600	3,400	4,900	6.7	38.2	55.1	8,900
283	Sarasota, FL	Total	400	4,200	4,700	4.3	45.2	50.5	9,300
284	Pascagoula-Moss Point, MS	Total	400	4,600	7,900	3.1	35.7	61.2	12,900
	COMBINED SMSAs								
285	El Paso, TX & Las Cruces, NM	Total	10,300	27,100	29,400	15.4	40.6	44.0	66,800
286	Greensboro-Winston-Salem High Point & Burlington, NC	Total	7,400	17,200	12,900	19.7	45.9	34.4	37,500

(continued on following page)

APPENDIX TABLE D. (continued)

			Children 6 months - 5 years						
			Number			Percent			
Rank	SMSA/Status*		Pre-1950	1950-69	1970-80	Pre-1950	1950-69	1970-80	Total
287	Fitchburg-Leominster & Pittsfield, MA	Total	6,400	3,300	3,200	49.6	25.6	24.8	12,900
288	Bangor & Lewiston-Auburn, ME	Total	6,300	2,700	2,600	54.3	23.3	22.4	11,600
289	Bristol & Meriden, CT	Total	5,400	4,100	2,200	46.2	35.0	18.8	11,700
290	Bismarck, ND & Grand Forks, MI	Total	5,100	6,000	8,800	25.6	30.2	44.2	19,900
291	Dubuque & Iowa City, IA	Total	5,000	4,000	4,900	36.0	28.8	35.3	13,900
292	Lawton & Enid, OK	Total	4,600	7,700	6,000	25.1	42.1	32.8	18,300
293	Laredo & Victoria, TX	Total	4,000	8,800	8,700	18.6	40.9	40.5	21,500
294	La Crosse, WI & Rochester, MN	Total	3,900	4,600	6,900	25.3	29.9	44.8	15,400
295	Little Rock & Pine Bluff, AR	Total	3,700	4,800	5,200	25.2	39.5	35.4	14,700
296	Bloomington, IN & Owensboro, KY	Total	3,700	5,700	6,300	23.6	36.3	40.1	15,700
297	Casper, WY & Great Falls, MT	Total	3,500	4,300	9,300	20.5	25.1	54.4	17,100
298	Midland & San Angelo, TX	Total	2,900	9,400	3,600	18.2	59.1	22.6	15,900
299	Topeka & Lawrence, KS	Total	2,800	4,800	6,200	20.3	34.8	44.9	13,800
300	Bryan-College Station & Sherman-Denison, TX	Total	2,400	5,900	5,700	17.1	42.1	40.7	14,000
301	Fort Walton Beach & Panama City, FL	Total	2,000	9,100	9,700	9.6	43.8	46.6	20,800

*Status: Inside Central City and Outside Central City

APPENDIX E

LEAD-CONTAMINATED SOIL CLEANUP
DRAFT REPORT*

P.L. Ciriello and T. Goldberg
Region I
U.S. Environmental Protection Agency,
Boston, MA

March 27, 1987

---

*This report is reproduced in its entirety because of its value to the issue of childhood lead exposure abatement strategies. The opinions and conclusions presented in this appendix are not necessarily those of ATSDR or the authors of the Report to Congress.

SUMMARY

Lead contaminated soil is ubiquitous in urban areas throughout the United States. It appears to be an especially serious problem in older cities with large numbers of homes with lead-based exterior paint. Public health officials are primarily concerned with the impacts of soil lead exposure on children ages 5 months to 6 years.

Children are exposed to the lead in soil through ingestion of the surface soil during play activity and inhalation of the soil when it becomes part of the dust in the air. The dust and soil is brought indoors on the clothing and shoes of adults and children and through open windows and becomes integrated in the indoor dust. Many children ages 6 months to 12 years engage in hand to mouth activity, which can lead to ingestion of large amounts of lead contaminated soil and dust.

In urban areas the major source of lead in contaminated soil is lead-based paint which has either been scraped off or has chipped off and weathered from the sides of buildings. Another less important source of lead is from auto exhaust. Lead smelters can also be a major source of soil contamination, however, these are uncommon in urban areas.

At this time, EPA has not set a lead standard for soil. In this report, we propose a priority action level of 1,000 ppm. This is based on a survey of action levels at several Superfund sites, an EPA biokinetic model, the CDC's policy, and the temporary standard in Minnesota. While we believe that a greater margin of safety would be achieved with an action level of 500 ppm, we think it is necessary to set priorities for remedial activity. The 1,000 ppm action level provides guidance for such priority setting.

This report evaluates five proposed remedial alternatives for lead contaminated soil in residential urban areas. These are: A) Removal and disposal of top soil offsite, covering with clean soil, and revegetation; B) Removal and disposal of top soil onsite, covering with clean soil, and revegetation; C) Covering soil with uncontaminated top soil and revegetation; D) Removal, decontamination, placement onsite, and revegetation; E) Rototilling soil and revegetation.

To analyze the impacts of these remedial alternatives, we characterized a typical site and qualitatively compared their public health impacts and costs. If the alternative has been proposed or implemented either at Superfund sites or other areas with lead contaminated soil, we described the experience and, if possible, the results. In our survey of remedial actions at different sites, we could find no example in which the agency responsible for the action followed up with a study of the impact on childhood blood lead levels.

The report concludes that: 1) excavation and decontamination is too costly, technically inappropriate and creates additional public health problems; 2) the impacts of capping, rototilling, and excavation and onsite disposal are uncertain in the long term because the contaminated soil remains onsite. These alternatives may be more appropriate for sites with soil lead less than 1,000 ppm. 3) Excavation with a study of the impacts on childhood blood lead levels, and offsite disposal provides the highest degree of protection to residents. However, the costs and safety of offsite disposal remains an important question. If disposal in a lined landfill is required by state law, the costs of this alternative will increase substantially. We recommend additional research into alternatives to landfill disposal.

The report concludes that effective remedial action at residential urban sites for lead contaminated soil requires an extensive public education program.

DRAFT

REMEDIAL ALTERNATIVES

INTRODUCTION

The purpose of this review is to describe and evaluate viable remedial alternatives for lead contaminated soil in urban residential environments. The first section provides an overview of the lead in soil problem encountered in a city like Boston, Massachusetts as a basis for analyzing 5 possible response actions. The assumptions used by the authors are presented in the overview section.

In the second section each alternative is examined. This section contains a brief description followed by a short analysis of public health effects and costs. In the section, we compare the alternatives.

We evaluate the following remedial alternatives for cleanup:

A) Removal and disposal of top soil offsite and revegetation;
B) Removal and disposal of top soil onsite and revegetation;
C) Covering soil with uncontaminated top soil and revegetation;
D) Removal, decontamination, placement onsite, and revegetation;
E) Rototilling soil and revegetation.

No action is an alternative, but it is not included in this analysis. Although we are concerned about exterior source control measures, interior deleading, interior dust control and recontamination of environments in the future, we do not evaluate them in this paper.

EPA has little experience addressing lead in soil problems in urban residential areas. * * * Since this experience is limited to industrial and commercial contamination sites and provides little quantitative information on the public health impacts, our comparison of alternative response actions is qualitative.

I. OVERVIEW--LEAD IN SOIL

The primary goal of any lead in soil remedial action is to eliminate the health hazards to children ages five months to six years from exposure to lead contaminated soil. According to recent studies, in areas where there are no emission point sources, the major sources of contamination are lead based paint which has flaked off the sides of buildings and to a lesser degree lead from gasoline.

In urban areas of extreme contamination, lead-based paint appears to be the major source (City of Boston, Department of Health and Hospitals, 1985 and Minnesota Pollution Control Agency, February 1987). There is some evidence indicating that a concentration gradient exists around residences. At locations where excessive lead in soil is found, the topsoil within six feet of the building's foundation usually contains the highest concentrations.

According to recent studies in Boston, in areas where large numbers of children have elevated blood lead levels and reside in housing with flaking paint, the average soil concentrations near the foundation is 2,000 ppm (City of Boston, Department of Health and Hospitals, 1985),

To facilitate regulatory and corrective activities, state and federal officials have been investigating the development of a soil lead standard (Minnesota Pollution Control Agency, February 1987). The EPA has not set a standard for lead contaminated soil. However, the Agency has conducted some research to evaluate the risks associated with proposed standards and some additional work is currently underway. According to a recent survey of available studies, blood lead levels can increase from 0.6 μg/dl to 9 μg/dl for every 1,000 ppm increase in the soil and dust lead level (Duggan and Inskip, 1985).

Using the EPA's Biokinetic/Integrated Uptake Model, Region I developed acceptable soil lead levels for Boston based upon an assessment of human exposure to lead in air. This model is described in detail in the Office of Air Quality Planning and Standards staff report on lead (OAQPS, February 1986).

The Massachusetts Special Legislative Commission on Lead Poisoning Prevention recently proposed legislation requiring the state's environmental agencies to establish a lead in soil standard 180 days after the passage of proposed legislation (Special Legislative Commission on Lead Poisoning Prevention, 1987).

In Minnesota, a draft report by the state's Pollution Control Agency examined the literature on the relationship between soil lead levels and blood levels. They report that there is inconclusive evidence on the hazards at 500 ppm or less. The experts consulted for the report agreed that 800 ppm and greater appear to pose a significant risk (Minnesota Pollution Control Agency, 1986). The Minnesota legislature has set an interim standard of 1,000 ppm and has asked the Pollution Control Agency to set a permanent standard for soil lead by 1988.

The CDC has stated that soil lead levels of 500 to 1,000 ppm contribute to increasing the blood lead levels in children above the background (Centers for Disease Control, 1985). In our survey of action levels at several Superfund sites, we found a range of 500 to 1,000 ppm with an average of 850 ppm.

Assumptions

We propose a priority soil lead action level for undertaking remedial steps of 1,000 ppm or greater (Duggan and Inskip, 1985; Centers for Disease Control, 1985). However, action may be necessary at 500 ppm and above. This is supported by the recommendations of the CDC and experience at Superfund sites.... However, due to the ubiquitous extent of the problem, we believe that incremental problem solving is necessary. We propose starting with soils that exceed 1,000 ppm in areas where contact is likely.

In this report, we evaluate all remedial alternatives on the basis of whether they reduce the soil lead content to below 500 ppm. If the soil lead is lowered to this level, we assume that the public health impacts will be minimized.

The analysis in this report is based upon a hypothetical site, which has a three story residence with soil lead levels of 2,000 ppm for several feet of surrounding area. A typical building dimension for urban homes in Boston is 60'x30'.

A visual survey of the neighborhoods with lead in soil problems in Boston revealed that the front side of many homes are situated on a sidewalk. The remaining three sides are often bordered by small patches of unvegetated ground. Because of density and development there often is less than 10 feet between homes. However, for this analysis we will assume that all four sides of the residence are bordered by soil for the purposes of consistency with other studies (City of Boston, Department of Health Hospitals, 1985).

We assume that the highest levels of lead in soil are within the first six feet of the foundation and drop off in the next six feet. This is based on sampling conducted at residences in Boston and Minnesota (Tom Spittler, personal communications, January, 1987; and Minnesota Pollution Control Agency, February 1987). Therefore, we will assume that seven feet on each of four sides of the house must be addressed.

Under these assumptions the total area for remedial action would be approximately 1,456 square feet.

We assume that the soil may be contaminated to a depth of three inches. The soil samples collected in Boston reveal that the top one inch contains the highest levels of lead and the concentration drops off significantly as one digs deeper (Spittler, personal communications, January 1987). The remedial alternatives described here are designed to dilute or remove at least the top three inches of soil. Because site conditions may differ, field examinations at each one should be conducted prior to any action.

Public Health Concerns

The major public health concerns for all remedial alternatives are, 1. potential occupational exposure and exposure of nearby residents during remedial action, 2. control of the sources of contamination, and 3. the long term integrity of the action. Another public health concern is the ability to remove the hazard, which is addressed in this report's discussion of individual remedial alternatives. Exposure during the remedial action can be controlled, but source control and long term control of the conditions of the site are not easily controlled.

1. Occupational adult exposure to lead during implementation of the remedial alternatives could be mitigated by careful planning and monitoring. This could include procedures such as those developed by the Massachusetts Port Authority in their lead in soil clean up program.

In general, these occupational health measures would involve basic efforts such as reducing dust during cleanup through wetting the soil and equipment; keeping nearby residents, especially children, away from the site; requiring workers to eat offsite and change their outer clothing before returning to their home. To test the adequacy of industrial hygiene techniques to control exposure during remedial action, a monitoring program should be established.

2. In the absence of source control, the long term effectiveness of remedial action is unknown. There are major uncertainties concerning the rate

of lead redeposition into soil at residential sites both from auto exhaust and from lead-based paint peeling off the sides of nearby buildings.

3. All remedial alternatives proposed in this report involve seeding or resodding the sites. This is proposed to minimize erosion and to provide some dust control. However, we are concerned about whether residents will maintain the revegetated areas. Furthermore, the density of structures creates large shaded areas which may not provide enough sunlight to support vegetation.

If sites are not maintained, there may be increased amounts of dust exposure at the site, and over time the original subsurface soil may be exposed. In these cases, shade tolerant groundcover or compacted soil may be the best option. There are also uncertainties concerning the long term maintenance of the surface soil at urban sites which can be significant for nonexcavation alternatives.

In the discussions of each remedial alternative, we describe the specific public health concerns that are expressed qualitatively and are based on the experience at either Superfund sites or other lead in soil clean up programs. In the conclusions of the report, we compare these concerns.

Cost Analysis

The cost analysis in this review provides a perspective on the relative costs of each alternative. These cost estimates are based on research presented in a report by Steve Nicholas, "Dealing with Lead-Contaminated Soil in Boston" which was written for a masters program at the Kennedy School of Government (Nicholas, 1986). The figures presented here modified the estimates in Nicholas' report by reducing the labor time to those estimated by the City of Boston and verified in this project.

The cost estimates do not include the costs of analyzing soil samples, administering the program, public education efforts, follow-up screening and soil testing, and other administrative costs associated with lead remedial action. This analysis also does not include costs of relandscaping an area. These additional costs are important but they are constants. The cost analysis does not include the expenses associated with maintaining a site after remedial action. These costs could vary depending on the remedial alternative. The costs estimates are based on information gathered in 1986 and are presented in 1986 dollars.

II.REMEDIAL ALTERNATIVES

A. Soil Removal, Disposal Offsite, and Revegetation

Procedure

Excavate approximately three inches of top soil. Removing three inches of topsoil from this space would produce approximately 13.5 cubic yards of soil. The soil is wetted down before removal to minimize dust. It is excavated by hand with shovels and loaded onto a truck and taken to an appropriate disposal site. The site is regraded with three inches of clean loam, and sod/grass seed/groundcover is sown to stabilize the site. The replacement loam is tested to ensure that it has an acceptably low lead content.

Once excavation and regrading are complete, the site will be tested to make sure that the top three inches of soil are below 500 ppm lead.

There are several disposal options for the contaminated soil. Right now the most viable options are disposal at either lined or unlined landfills. Arthur D. Little (ADL), a Cambridge-based consulting firm, is conducting a study for the Massachusetts Port Authority to examine these and other options, such as disposal in large pits as is currently done with fly ash, use as fill in large construction sites with deep foundations, or mixing the soil with cement and asphalt for use in construction of roads (Pat Hynes, personal communications, 1987).

Identifying an environmentally acceptable disposal option for this remedial alternative is critical. Acceptable disposal options may have a substantial impact on the costs of implementing this alternative.

Discussion

This discussion is based on the experience of the Massachusetts Port Authority (MASSPORT) soil clean up program under the Tobin Bridge in Chelsea, Massachusetts. This program was started in 1977 when MASSPORT initiated a study of the soil lead levels in the residential area adjacent to and under the Bridge.

MASSPORT's research found that contamination of soil was the result of flaking paint from nearby residences, fine dust particles from sand blasting the bridge, and gasoline exhaust from bridge traffic. After taking a number of steps to improve the soil lead conditions at several sites directly under and adjacent to the bridge, MASSPORT initiated a program of excavating all the soil within one block on either side of the bridge.

Last summer MASSPORT excavated the soil around a public school in the area to assess the effectiveness of their proposed remedial action. The Authority removed 6 inches of topsoil by small tractor, called a Bobcat®, replaced it with 6 inches of clean loam from offsite, and regraded and resodded the site. They removed the soil from the site each day by truck and disposed of it in a lined landfill on the property of the contractor involved in excavation. The site was relandscaped according to plans developed jointly by MASSPORT and school officials.

To control occupational exposures, the contractor was required to follow a health and safety plan, which was based on OSHA guidelines and written by a private industrial hygienist. This plan is outlined in the report, "Plan and Instructions for Safety, Removal of Lead Contaminated Soil in the Tobin Bridge Community Project" (MASSPORT, 1985).

One major form of protection was wetting soil prior to removal to minimize dust dispersal. A major source of dust and potential exposure that required control measures was the soil that became imbedded in the wheels of the bobcat. This too was controlled by periodic wetting.

In addition, the health and safety plan required workers to eat offsite, to wash before eating, and to remove their outer work clothes before going home. To protect nearby residents, MASSPORT required the contractor to post notices in the neighborhood warning people not to come onto the site during construction.

According to MASSPORT officials, monitoring of lead in air during soil removal indicated that the levels were not elevated. The air lead OSHA standard was not exceeded at any point during the project (Pat Hynes, Director of Environmental Programs, MASSPORT, personal communications, December 1986).

In another case, emission from a lead smelter caused serious soil contamination in a nearby Dallas, Texas neighborhood. Within a half mile radius of the plant the soil lead levels were 1,000 to 2,500 ppm. A consent agreement with EPA required the responsible firm to eliminate hazards due to lead contaminated soil. EPA and state and local health officials cooperated in the development of a remedial plan. It required removal of the top six inches of soil; replacement with clean soil, and revegetation of the site (U.S. EPA, 1985). Unpaved driveways had three inches of soil removed and replaced with limestone.

These procedures were carried out in 1985. Unfortunately for the purposes of this report, the soil removal program was not followed by an examination of the impacts on the blood lead levels of children in area homes during the subsequent year. Many of the health and safety procedures required at the MASSPORT site to minimize exposure during excavation were also required at the Dallas site.

Consequences

Since the top three inches of contaminated soil contains the high lead concentrations, removal of three inches of top soil, disposal offsite, capping with new topsoil and revegetation should eliminate the hazard for children. This alternative would meet the goal of reducing the soil lead content to below 500 ppm for the short term.

The MASSPORT project is still too recent to rely on their example for information about long term effectiveness. Officials plan to monitor the site annually to assess the rate of redeposition and whether soil removal provides a permanent solution.

The major public health concern for this alternative is potential hazards at the site of disposal. The methods of offsite disposal present some potential unknown risks for the long term. Public health impacts at the disposal site will be regulated by the state environmental protection and public health authorities. At this time, we assume that disposal at a landfill which is lined is always a safer alternative than disposal at an unlined landfill. However, it is unclear whether the additional degree of safety is required to protect the environment and public health.

There is a debate among environmental health officials over the basis for determining the safety of land-based disposal options. On one side, the Superfund statute identifies lead as a hazardous substance and requires that disposal be based on the results of EP toxicity tests. EP toxicity tests conducted by Region 1 Environmental Services Division reveal that lead does not leach into groundwater under normal conditions until the levels reach approximately 15,000 ppm.

On the other side, some argue that there are uncertainties about long-term environmental health impacts associated with land-based disposal, and EP toxicity testing is not an adequate basis for decision-making. The major concern about disposal in an unlined landfill is the possibility for the lead to leach from contaminated soil if it is exposed to acids from the waste.

If lead contaminated soil is used to cap either lined or unlined landfills, one long term public health concern is the ability of the landfill owner to keep children from getting onto the site and to secure the site so the soil does not erode or blow offsite.

Costs

Labor for removing soil (estimated time, 12 hrs. @ $18/hr.): $216.00
Labor for spreading new soil and revegetating (estimated at 12 hrs.): $216.00
Loam: $215.00
Seed: $10.00
Transportation of Soil Offsite: $404.00
Disposal of Soil Offsite in lined landfill: $270.00
Disposal of Soil Offsite in unlined landfill: $40.00
Total for Disposal in lined landfill: $1,357.00
Total for Disposal in unlined landfill: $1,127.00

B. Removal of Soil and On-Site Burial and Revegetation

Procedure

Excavate approximately three inches of topsoil. To reduce reentrainment of the dust during excavation, the soil should be sprayed with water during the removal process. Soil is removed by hand with shovels and moved by wheelbarrows to the onsite disposal area. The onsite disposal area is a pit that is deep and wide enough to hold the excavated material and a two foot cap of clean soil up to grade. The pit is dug either by hand or with the assistance of a small tractor. The excavated area is regraded with three inches of clean soil and reseeded or resodded. The replacement loam is tested to ensure that it has an acceptably low lead content.

Discussion

The Boston Office of Environmental Affairs excavated and buried contaminated soils onsite at several residences in Boston. On site disposal is considerably less expensive than excavation and offsite disposal (Ron Jones, lead program director, personal communications, February, 1987). It has been used as a "home grown" alternative for residents interested in taking independent action.

Consequences

This remedial alternative would have the same positive short term effects on mitigating exposure for children to topsoil contaminated with lead as excavation and offsite disposal because topsoil lead content is reduced to below 500 ppm. The long term effectiveness of this alternative, however, is even less certain than that for offsite disposal. There is still a possibility of exposure because the soil has not been permanently removed from the site.

A major public health concerns for this proposal is the possibility that the soil will be dug up sometime after the remedial action has taken place. Children could dig up the soil during their play activity, the freeze and thaw cycle could bring the lead contaminated soil to the surface over a period of years, and construction or gardening could uncover the soil.

Costs

Labor for removing soil (estimated time 12 hrs. @ $18.00/hr): $216.00
Labor for spreading new soil and revegetation (estimated at 12 hrs.): $216.00
Labor for digging pit and burying soil (estimated time 8 hrs.): $144.00
Loam: $205.00
Seed: $10.00
Total: $791.00

C. Cover with Uncontaminated Topsoil and Revegetation

Procedure

Transport clean soil to the site and rake it into place. Cap the site with 6 inches of loam. Replacement soil is tested to ensure that it has an acceptably low lead content. The soil is moved on to the site by hand with shovels and wheelbarrows and the "capped" area is reseeded or resodded. Deed restrictions are attached to the property to ensure that the integrity of the cap is preserved and maintained.

Discussion

Site capping has been proposed as a remedial alternative for lead contaminated soil by the Minnesota Pollution Control Agency (MPCA) and by EPA at the Industri-plex Superfund site in Woburn, Massachusetts. The MPCA proposed capping sites with low to moderate soil lead levels as one remedial alternative which landowners could use. The Agency does not specify a cut off lead level for low or moderately contaminated soil. They recommended adding two inches of clean soil at these sites.

We believe that a two inch cap would not be adequate for residential sites with soil contaminated with 1,000 ppm PB or greater. A two inch cap could be quickly eroded and could be easily penetrated during normal child play activity.

At the Industri-plex hazardous waste site in Woburn, Massachusetts, EPA decided to cover areas with lead contaminated topsoil and revegetate. The Agency will add 30 inches of material to place the contaminant below the freeze/thaw zone and mitigate the likelihood that contaminated soil will be carried to the surface by this process. The thirty inch cap would also eliminate the potential for direct contact.

At this site, EPA selected an action level of 600 ppm lead. Lead was found in excess of 1,000 ppm in soil covering about one half of the site.

There were, however, other heavy metal and volatile organic compounds on the site, which were of major concern in selecting the remedial action. As a result, removal of the soil was not selected because of the presence of buildings on the site and the potential for creating serious odors if piles of contaminated hides were disturbed. In areas surrounding the buildings, it would not be feasible to add thirty inches of soil. In these areas, EPA will probably remove the soil and replace it with clean fill or place asphalt over the soil. At publication of this review, a final decision on this has not been made.

The analogy between the Industri-plex site and the situation in urban areas is limited. Most of the lead contaminated soil in urban areas is close to buildings where it would not be feasible to add thirty inches of soil. To add a cap which is much smaller than this, would risk putting the lead contaminated soil in the freeze and thaw zone.

We propose a six inch cap for lead contaminated soil because it would not be as easily penetrated by child activity or as quickly eroded by rain, wind, and human activity as a two inch cap. There are uncertainties, however, concerning the effects of the freeze and thaw cycle on the integrity of a 6 inch cap. According to Alan Dusault from the Solid Waste Division of the Massachusetts Department of Environmental Quality Engineering, this should not be a problem for the soil types in Boston. However, soil in other areas may be susceptible to the impact of freeze and thaw. If capping is selected as a remedial alternative, the effects of this cycle on the cap should be evaluated.

Consequences

This remedial alternative could meet the immediate term goal of reducing surface soil lead content to below 500 ppm. However, a major public health concern with this alternative is its long term effectiveness. At many residential sites, it may be impossible to retain a grass cover either because of negligence by the landowner or because of a lack of sunlight. If the soil is bare there is a greater likelihood of erosion and direct contact with children.

In addition, it would be impossible to control the children's play activities associated with digging up surface soil. This activity creates the likelihood of exposure. There are similar public health concerns for protection of work crews and nearby residents in this remedial alternative as for the others. However, the surface soil will not be disturbed which could lesson the possibility of direct contact between these adults and the lead in the soil.

At an industrial hazardous waste site, EPA can require institutional controls after remedial action. One commonly used control is restrictions on the property's deed. Such restrictions may not be feasible to implement at residential sites. In the absence of institutional control, it would be impossible for the future activities on the site to be restricted.

Costs

Labor for spreading new soil and revegetation (estimated time 12 hrs. @ $18/hr): $216.00

Loam: $410.00

Seed: $10.00

Total: $726.00

D. Removal, Decontamination, Placement Onsite, and Revegetation

Procedure

Three inches of top soil would be removed and transported to a nearby facility for chelation. Lead can be separated from soil using EDTA as a chelating agent in an aqueous solution. The chelation process removes all other minerals and soil nutrients. To make the soil useful for growing vegetation, the necessary inorganic and organic elements would have to be replaced. This would be done before placing the decontaminated soil back onsite. The site would be revegetated.

Discussion

EPA did a preliminary analysis of this alternative for lead contaminated soils found in urban areas. Their research revealed that the procedure removes approximately 99% of the lead.

The Agency decided not to pursue a full investigation of this alternative for several reasons: 1) there would be residue of chelating agent present in the soil, which would cause the residue of lead in the soil to be highly mobile along with all the other materials in the soil. These mobile elements would easily leach into groundwater; 2) the size and cost of the facility necessary to process the soil and cost would be inappropriate for an urban setting; 3) discussions with EPA staff at demonstration sites indicated that the process needs more development before a demonstration is attempted; 4) the necessity of replacing all the important nutrients back into the soil before it can be reused would substantially increase the cost; and 5) experience in other contexts indicates that there are unresolved problems associated with recycling the EDTA agent. Soil chelation creates a highly toxic lead residue, which is extremely mobile and must be disposed in a contained cell.

Consequences

This remedial alternative could achieve the public health goal of reducing the soil lead level to below 500 ppm. However, there are major long term public health concerns associated with this action, including groundwater contamination and the toxicity of the EDTA residue after processing. Unless this alternative is accompanied by source control measures, any lead that becomes deposited on the soil will be highly mobile and easily leached.

Costs

At this time, we do not have accurate cost estimates for this remedial alternative. Based on a preliminary analysis, the costs of decontamination would make this alternative significantly more expensive than other alternatives. At this time, decontamination would require building a facility for chelation near the excavated areas, which would be major expense and pose substantial logistical problems.

E. Rototill and Revegetate

Procedure

Rototill the lead contaminated soil area and seed it with grass. A standard rototill machine digs approximately 18 inches deep into the soil. By repeated rototilling, one can approach a thorough mixing of the top and bottom soil.

Prior to rototilling an area, the soil would be tested to ensure that the soil below the surface had lower lead levels than on the surface. This testing should include analyzing soil depth profiles throughout the site especially near the foundation. To analyze whether rototilling a site would achieve a reduction of topsoil lead less than 500 ppm, one could calculate depth profiles that could achieve this under the conditions at typical sites.

Discussion

In a recent draft report, the Minnesota Pollution Control Agency (MPCA) proposed rototilling soil in areas with low to moderate soil lead levels, and with children who have elevated blood lead levels and access to the site. This is one of three alternatives MPCA may make available to the site owner. The report does not define the range of lead contamination considered by the Agency to be low or moderate. They argue that this will depend on the state's soil lead standard. The Agency believes that this alternative would be an inexpensive and feasible way to reduce exposure to lead in topsoil. They do not believe it is adequate in areas with high lead in soil contamination and high childhood blood lead levels. In the MPCA report, the authors do not analyze the dilution factor associated with this alternative to determine how effective it would be in minimizing exposure (MPCA, 1986). As far as we know, rototilling has not been demonstrated in Minnesota or elsewhere.

Consequences

Rototilling a site may or may not achieve the public health goal of reducing the lead in topsoil to below 500 ppm. If there is relatively high lead content of the topsoil and subsurface soil, this alternative may not be appropriate. In areas with high lead content of the top inch and very low lead levels in subsurface soil, this alternative could reduce the lead in top soil substantially. This alternative would not eliminate lead from the surface soil. It is likely that topsoil would have to be capped to cover the indigenous rototilled material because subsurface soils are inappropriate as a growing medium.

Costs

Labor for rototilling site (estimated time, 12 hrs. @ $18/hr.): $216.00
Labor for reseeding site (estimated time 2 hrs.): $36.00
Equipment rental (small rototiller rented for 2 days @ $37/day): $74.00
Seed: $10.00
Total: $336.00

III. CONCLUSIONS

Selecting Remedial Alternatives

In the absence of a soil lead standard, we have proposed a soil lead action level of 1,000 ppm or greater in urban areas. This is based on our review of EPA's biokinetic model, CDC's policy statement, action levels for superfund sites, and the available literature on the impacts of lead in soil on childhood lead uptake.

We recommend that remedial action at sites with soil lead levels of 1,000 ppm or greater be undertaken regardless of whether there are children living in the site's residence. Urban populations tend to be mobile and children could move into a residence at any time. However, if a choice of using limited resources to cleanup a site with children up to six years of age or to cleanup a site without children, we believe that the site with children should be given higher priority.

All of the remedial alternatives proposed in this report are technically feasible (See Table E-1 for a comparison of remedial alternatives). However, we have eliminated excavation and off site decontamination from serious

TABLE E-1. SUMMARY OF REMEDIAL ACTION ALTERNATIVES

Alternative Description	Estimated Costs (1986 Dollars)	Public Health Concerns	Prior Experience	Technical Concerns	Institutional Concerns	Comments
EXCAVATION						
Soil removal, disposal and revegetation						
-Disposal in lined landfill	$1,240-1,357	Reduces lead in exposure; no human contact with contaminated soil	MASSPORT Cleanup in Chelsea, MA	Proven technology; easy to implement	Availability of lined landfill; state and local restrictions	
-Disposal in unlined landfill	$1,127	Reduces lead in soil exposure; no human contact with contaminated soil. Concerns about exposure at disposal site	Dallas, TX	Proven technology; easy to implement	Availability of landfill sites; state and local restrictions	
-Disposal on site	$791	Reduces lead in soil exposure. Long-term concerns about integrity of disposal - possibility for contaminated soil to be brought to the surface	Boston, MA	Proven technology; easy to implement. Concerns about availability of space for pit	Local regulations	
-Decontamination and placement on site	Not Available	99% removal of lead; concerns for increased mobility of lead resulting in groundwater contamination, and disposal of EDTA residue	--	Concerns for the efficiency of lead removal; staging facility near sites and replacement of soil nutrients. Complicated to implement	State and local restrictions	

(continued on following page)

TABLE E-1. SUMMARY OF REMEDIAL ACTION ALTERNATIVES

Alternative Description	Estimated Costs (1986 Dollars)	Public Health Concerns	Prior Experience	Technical Concerns	Institutional Concerns	Comments
CAPPING						
Placement of several inches of clean loam and revegetation	$726	Reduces lead in soil exposure. Concerns about long-term effectiveness-potential erosion and reentrainment of cap	Industri-plex SF site in Woburn, MA	Proven technology easy to implement	Concerns about restrictions on future use	May be most appropriate for soil lead levels less than 1,000 ppm
ROTOTILL						
Rototill and revegetation	$336	Reduces lead in soil exposure, but does not eliminate contaminated surface soil. Concerns about long-term effectiveness and exposure	Proposed in Minnesota	Easy to implement		May be most appropriate for soil lead levels less than 1,000 ppm

Notes:

The following are the uncertainties associated with all the remedial alternatives:

-source control and rate of redeposition
-long term maintenance of site
-resident's expectations for a "clean" environment
-compliance with state and local laws

consideration because it is too expensive and creates potential public health problems. There are major uncertainties concerning long term effectiveness of capping and rototilling because they do not involve removal of the contaminants from the site. They could provide some protection, especially for sites with soil lead levels below 1,000 ppm. Capping and rototilling could protect children in the short term from direct exposure to lead. We do not believe that they provide a permanent solution. Excavation and on site disposal presents similar questions as rototilling and capping.

We believe that excavation and off site disposal provides the greatest degree of protection for children on the site. However, disposal of the soil off site may be expensive. There is debate concerning the safety of disposing of lead contaminated soil in landfills. Whether the soil should be disposed of in lined or unlined landfills may depend on state and local regulations. If disposal in a lined landfill is required, the costs of remedial action will be greatly increased.

We encourage research into creative solutions to the problems associated with off site disposal. These include using the soil in asphalt batching, pits such as those used to dispose of fly ash, fill in construction sites, land reclamation, and closure caps for local landfills. Several of these alternatives are currently under investigation.

The selection of a remedial action will depend on the specific conditions of a site. Table E-2 presents a proposed decision-making matrix matching one set of site conditions and remedial alternatives. The axis with site conditions could be expanded to include many other parameters. This kind of matrix could be useful to environmental and public health officials involved in soil cleanup.

The assumption of remedial action for lead contaminated soil is that by reducing this potential source of exposure, one will observe a reduction in the lead in children's bodies. However, the experience in Boston, Chelsea, and Dallas provide no data to support this. We believe that in the design of remedial programs, it is important to collect information of the program's impact on reducing the blood lead levels of children. This data would be useful in the design of future remedial activities.

TABLE E-2. PROPOSED REMEDIAL ACTION DECISION-MAKING MATRIX

Remedial Action Soil Pb Content	Excavation Off-Site Disposal	Excavation On-Site Disposal	Capping	Rototill
0-500 ppm	X	X	X	X
501-1,000 ppm	X	X	X	X
1,000-2,000 ppm	X	?		
2,000 ppm +	X	?		

Notes:

The above matrix is a proposed model to use in a site evaluation. The matrix could assist the environmental health authorities to decide which remedial alternative is appropriate for the site's conditions.

Costs

The cost comparison presented in Table E-3 shows that the costs of all the remedial alternatives except for off sit disposal are within a close range. If local or state officials require disposal of excavated soil in a lined landfill, the costs of remedial action will increase substantially.

Public Education

In our conversations with officials at MASSPORT, they emphasized the importance of public education in any clean-up program. They have undertaken an extensive program of community meetings and one to one contact with residents. This has facilitated community awareness of the importance of the problem as well as cooperation in MASSPORT"S efforts. We suggest that a community education plan be integrated into any plan for remedial action at urban sites with lead contaminated soil.

TABLE E-3. COST COMPARISON FOR REMEDIAL ALTERNATIVES

	Excavation and Onsite Disposal *	Excavation and Offsite Disposal at Lined Landfill **	Excavation Disposal at Unlined Landfill ***	Excavation and Decontamination ****	Capping with 3 inches of Topsoil *****	Rototilling ******
Cost per site for removing 13.5 yard^3 topsoil (depth of 3 inches)	$791	$1,240-$1,357	$1,127	Not Available	$726	$336
Cost per yard^3	$59	$90-$100	$84	Not Available	$54	$23

*Estimate is based on labor and material costs reported in, "Dealing with Lead-Contaminated Soil in Boston" by Steven Nicholas. It assumes $18/hour labor total labor time/site of 32 hours, $205 for loam, and $10 for seed.

**Estimate is reported as a range. The lower value is based on estimates from MASSPORT. The upper estimate is based on the report by Steve Nicholas cited above. It assumes $18/hour labor, total labor time/site of 24 hours, $205 for loam, $10 for seed, $440 transportation costs, and $270 disposal costs.

***Estimate is based on the report by Steve Nicholas cited above. The difference between this estimate and the one for disposal at a lined site is cost of landfilling. It assumes $40 for cost of disposal.

****As reported in the text, these estimates are not available.

*****This estimate is based on the report by Steve Nicholas cited above. It assumes $18/hour labor, total labor time of 12 hours, $205 for loam, and $10 for seed.

******This estimate is based on a telephone inquiry to a rototill rental firm in Boston. The reported cost to rent a small machine per day is $37, and rental would be required for 2 days. The authors assume that labor time for rototilling would be 12 hours and revegetation would be 2 hours, costs $18/hour, and seed $10.

REFERENCES

1. Minnesota Pollution Control Agency. 1986. Draft Legislative Report to the Committee on Health and Human Services. St. Paul, Minnesota.

2. City of Boston, Department of Health and Hospitals, Office of Environmental Affairs. 1985. Boston Child Lead Poisoning: Request for Immediate Clean Up of Lead-Contaminated Soil in Emergency Areas. Boston, Massachusetts.

3. Duggan, M. and M. Inskip, 1985. Childhood Exposure to Lead in Surface Dust and Soil: A Community Health Problem. Public Health Review 13:1-59.

4. MASSPORT. 1985. Plan and Instructions for Safety Removal of Lead Contaminated Soil in Tobin Bridge Community Project. Unpublished, Boston, Massachusetts.

5. Minnesota Pollution Control Agency. 1986. Draft Legislative Report to the Committees on Health and Human Services. St. Paul, Minnesota.

6. Nicholas, Steve. 1986. Dealing with Lead-Contaminated Soil in Boston. A Policy Analysis Exercise for the John F. Kennedy School of Government. Unpublished. Cambridge, Massachusetts.

7. Special Legislative Commission on Lead Poisoning Prevention. 1987. The Continuing Toll, Lead Poisoning Prevention in the Commonwealth: Current Efforts and Future Strategies. Boston, Massachusetts.

8. U.S. EPA. 1985. Administrative Order on Consent, Docket No. CERCLA U1-5-83.

9. U.S. EPA. September, 1985. Superfund Record of Decision: Celtor Chemical, California.

10. U.S. EPA. March, 1986. Superfund Enforcement Decision Document: Pepper's Steel, Florida.

11. U.S. EPA. September, 1986. Superfund Record of Decision: Caldwell Trucking, New Jersey.

12. U.S. EPA. September, 1986. Superfund Record of Decision: Arcanum Iron and Metal, Ohio.

13. U.S. EPA. September, 1986. Superfund Record of Decision: Industri-plex, Woburn, Massachusetts.

APPENDIX F

FINAL AND PROPOSED NATIONAL PRIORITIES LIST WASTE SITES WITH LEAD AS AN IDENTIFIED CONTAMINANT

FINAL AND PROPOSED NATIONAL PRIORITY LIST (NPL) WASTE SITES WITH LEAD AS AN IDENTIFIED CONTAMINANT

EPA Region	State	Site Name	Location	Status	Hazard Ranking System Score
01	MA	Nyanza Chemical Waste Dump	Ashland	Final	69.22
01	MA	Shpack Landfill	Norton/Attleboro	Final	29.45
01	NH	Kearsarge Metallurgical Corp.	Conway	Final	38.45
01	RI	Davis (GSR) Landfill	Glocester	Final	38.89
02	NJ	Beachwood/Berkely Wells	Berkely Township	Final	42.24
02	NJ	Burnt Fly Bog	Marlboro Township	Final	59.16
02	NJ	Caldwell Trucking Co.	Fairfield	Final	58.30
02	NJ	CPS/Madison Industries	Old Bridge Township	Final	60.73
02	NJ	Imperial Oil/Champion Chemicals	Morganville	Final	33.87
02	NJ	Lang Property	Pemberton Township	Final	48.89
02	NJ	Lipari Landfill	Pitman	Final	75.60
02	NJ	NL Industries	Pedricktown	Final	52.96
02	NJ	PJP Landfill	Jersey City	Final	28.73
02	NJ	Price Landfill	Pleasantville	Final	71.60
02	NJ	South Brunswick Landfill	South Brunswick	Final	53.42
02	NJ	Syncon Resins	South Kearny	Final	43.43
02	NJ	Ciba-Geigy Corp.	Toms River	Final	50.33
02	NJ	Delilah Road	Egg Harbor Township	Final	49.33
02	NJ	Ewan Property	Shamong Township	Final	50.19
02	NJ	Florence Land Recontouring Landfill	Florence Township	Final	47.39
02	NJ	Ventron/Velsicol	Wood Ridge Borough	Final	51.38
02	NJ	Monitor Devices/Intercircuits, Inc.	Wall Township	Final	41.93
02	NY	Mercury Refining, Inc.	Colonie	Final	44.58
02	NY	Pollution Abatement Services	Oswego	Final	70.80
02	NY	Ramapo Landfill	Ramapo	Final	44.73
02	NY	Syosset Landfill	Oyster Bay	Final	54.27
02	NY	Johnstown City Landfill	Town of Johnstown	Final	48.36
02	NY	Volney Municipal Landfill	Town of Volney	Final	32.89
02	NY	FMC Corp. (Dublin Road Landfill)	Town of Shelby	Final	32.89
02	NY	North Sea Municipal Landfill	North Sea	Final	33.74
03	DE	Army Creek Landfill	New Castle County	Final	69.92
03	DE	Delaware Sand & Gravel Landfill	New Castle County	Final	46.60
03	DE	Harvey & Knott Drum, Inc.	Kirkwood	Final	30.77
03	DE	New Castle Steel	New Castle County	Final	30.40

(continued on following page)

FINAL AND PROPOSED NATIONAL PRIORITY LIST (NPL) WASTE SITES WITH LEAD AS AN IDENTIFIED CONTAMINANT

EPA Region	State	Site Name	Location	Status	Hazard Ranking System Score
03	DE	Tycouts Corner Landfill	New Castle County	Final	73.67
03	DE	Wildcat Landfill	Dover	Final	30.61
03	DE	E.I. DuPont (Newport Plant Landfill)	Newport	Proposed	51.91
03	MD	Southern Maryland Wood Treating	Hollywood	Final	34.21
03	MD	Aber Prov Ground-Michaelsville Landfill	Aberdeen	Proposed	31.45
03	MD	Woodlawn County Landfill	Woodlawn	Final	48.13
03	PA	Douglassville Disposal	Douglassville	Final	55.18
03	PA	Kimberton Site	Kimberton Borough	Final	29.44
03	PA	Mill Creek Dump	Erie	Final	49.31
03	PA	Osborne Landfill	Grove City	Final	54.60
03	PA	Resin Disposal	Jefferson Borough	Final	37.69
03	PA	Taylor Borough Dump	Taylor Borough	Final	30.94
03	PA	Dorney Road Landfill	Upper Macungie Two	Final	46.10
03	PA	Industrial Lane	Williams Township	Final	42.47
03	PA	Modern Sanitation Landfill	Lower Windsor Two	Final	33.93
03	PA	MW Manufacturing	Valley Township	Final	46.44
03	PA	Brown's Battery Breaking	Shoemakersville	Final	37.34
03	PA	Rohm and Haas Co. Landfill	Bristol Township	Proposed	48.51
03	PA	Keystone Sanitation Landfill	Union Township	Final	33.76
03	PA	Revere Chemical Co.	Nockamixon Township	Final	31.31
03	PA	C & D Recycling	Foster Township	Final	43.92
03	VA	U.S. Titanium	Piney River	Final	34.78
03	VA	Love's Container Services Landfill	Buckingham County	Proposed	40.71
03	VA	Saunders Supply Co.	Chuckatuck	Proposed	55.57
03	VA	C&R Battery Co., Inc.	Chesterfield County	Final	46.44
03	VA	H&H Inc. Burn Pit	Farrington	Proposed	39.04
04	AL	Olin Corp. (McIntosh Plant)	McIntosh	Final	39.71
04	AL	Stauffer Chem (LeMoyne Plant)	Axis	Final	32.34
04	AL	Alabama Army Ammunition Plant	Childersburg	Final	36.83
04	AL	Interstate Lead Co. (ILCO)	Leeds	Final	42.86
04	FL	Alpha Chemical Corp.	Galloway	Final	43.24
04	FL	Davie Landfill	Davie	Final	57.86
04	FL	Gold Coast Oil Corp.	Miami	Final	57.80

(continued on following page)

FINAL AND PROPOSED NATIONAL PRIORITY LIST (NPL) WASTE SITES WITH LEAD AS AN IDENTIFIED CONTAMINANT

EPA Region	State	Site Name	Location	Status	Hazard Ranking System Score
04	FL	Kassauf-Kimerling Battery	Tampa	Final	53.42
04	FL	Miami Drum Services	Miami	Final	53.56
04	FL	Munisport Landfill	North Miami	Final	32.37
04	FL	NW 58th Street Landfill	Hialeah	Final	49.43
04	FL	Pickettville Road Landfill	Jacksonville	Final	42.94
04	FL	Sapp Battery Salvage	Cottondale	Final	47.70
04	FL	Schuylkill Metals Corp.	Plant City	Final	59.16
04	FL	Whitehouse Oil Pits	Whitehouse	Final	52.58
04	FL	Sixty-Second Street Dump	Tampa	Final	49.09
04	FL	City Industries, Inc.	Orlando	Proposed	32.00
04	FL	Peak Oil Co./Bay Drum Co.	Tampa	Final	58.15
04	FL	Montco Research Products, Inc.	Hollister	Proposed	29.44
04	FL	Petroleum Products Corp.	Pembroke Park	Final	40.11
04	GA	Monsanto Corp. (Augusta Plant)	Augusta	Final	35.65
04	KY	A.L. Taylor (Valley of Drums)	Brooks	Final	17.68
04	KY	Distler Farm	Jefferson County	Final	34.62
04	KY	Newport Dump	Newport	Final	37.63
04	KY	Smith's Farm	Brooks	Final	32.69
04	MS	Flowood Site	Flowood	Final	8.27*
04	NC	Chemtronics, Inc.	Swannanoa	Final	30.16
04	NC	Jadco-Hughes Facility	Belmont	Final	42.00
04	SC	Independent Nail Co.	Beaufort	Final	57.90
04	SC	Kalama Specialty Chemicals	Beaufort	Final	57.90
04	SC	Wamchem, Inc.	Burton	Final	47.70
04	SC	Palmetto Recycling, Inc.	Columbia	Final	29.46
04	TN	Lewisburg Dump	Lewisburg	Final	33.45
04	TN	Milan Army Ammunition Plant	Milan	Final	58.15
05	IL	Byron Salvage Yard	Byron	Final	33.93
05	IL	Kerr-McGee (Sewage Treat Plant)	West Chicago	Proposed	35.20
05	IL	NL Industries/Taracorp Lead Smelt	Granite City	Final	38.11
05	IL	Petersen Sand & Gravel	Libertyville	Final	38.43
05	IL	Sheffield (U.S. Ecology, Inc.)	Sheffield	Proposed	29.49
05	IL	Joliet Army Ammu Plant (M Area)	Joliet	Final	32.08
05	IL	Joliet Army Ammu Plant (LAP Area)	Joliet	Proposed	35.23

(continued on following page)

FINAL AND PROPOSED NATIONAL PRIORITY LIST (NPL) WASTE SITES WITH
LEAD AS AN IDENTIFIED CONTAMINANT

EPA Region	State	Site Name	Location	Status	Hazard Ranking System Score
05	IN	MIDCO I	Gary	Final	46.44
05	IN	MIDCO II	Gary	Final	30.16
05	IN	Poer Farm	Hancock County	Final	37.38
05	IN	Waste, Inc., Landfill	Midland City	Final	50.63
05	MI	Berlin & Farro	Swartz Creek	Final	66.74
05	MI	Kentwood Landfill	Kentwood	Final	35.39
05	MI	Rose Township Dump	Rose Township	Final	50.92
05	MI	Speigelberg Landfill	Green Oak Township	Final	53.61
06	OK	Tar Creek (Ottawa County)	Ottawa County	Final	58.15
06	OK	Sand Springs Petrochemical Complex	Sand Springs	Final	28.86
06	TX	Bio-Ecology Systems, Inc.	Grand Prairie	Final	35.06
06	TX	Highlands Acid Pit	Highlands	Final	37.77
06	TX	Lone Star Army Ammunition Plant	Texarkana	Final	31.85
06	TX	Sheridan Disposal Services	Hempstead	Proposed	30.67
07	IA	Vogel Paint & Wax Co.	Orange City	Final	31.45
06	OK	Tar Creek (Ottawa County)	Ottawa County	Final	58.15
06	OK	Sand Springs Petrochemical Complex	Sand Springs	Final	28.86
06	TX	Bio-Ecology Systems, Inc.	Grand Prairie	Final	35.06
06	TX	Highlands Acid Pit	Highlands	Final	37.77
06	TX	Lone Star Army Ammunition Plant	Texarkana	Final	31.85
06	TX	Sheridan Disposal Services	Hempstead	Proposed	30.67
07	IA	Vogel Paint & Wax Co.	Orange City	Final	31.45
07	KS	Johns' Sludge Pond	Wichita	Final	35.94
07	KS	Cherokee County	Cherokee County	Final	58.15
07	KS	Big River Sand Co.	Wichita	Final	32.56
07	KS	National Industrial Environ Serv	Furley	Proposed	39.59
07	KS	Strother Field Industrial Park	Cowley County	Final	33.62
07	MO	Quality Plating	Sikeston	Final	40.70
07	MO	Wheeling Disposal Service Co. Lf	Amazonia	Proposed	29.85
08	CO	California Gulch	Leadville	Final	55.84
08	CO	Uravan Uranium (Union Carbide)	Uravan	Final	43.53
08	MT	Anaconda Co. Smelter	Anaconda	Final	58.71
08	MT	Milltown Reservoir Sediments	Milltown	Final	43.78
08	MT	Silver Bow Creek/Butte Area	Silver Bow/Deer Lodge	Final	63.76
08	MT	East Helena Site	East Helena	Final	61.65

(continued on following page)

FINAL AND PROPOSED NATIONAL PRIORITY LIST (NPL) WASTE SITES WITH LEAD AS AN IDENTIFIED CONTAMINANT

EPA Region	State	Site Name	Location	Status	Hazard Ranking System Score
08	UT	Sharon Steel (Midvale Tailings)	Midvale	Proposed	73.49
08	UT	Portland Cement (Klin Dust 2 & 3)	Salt Lake City	Final	54.40
08	UT	Olson/Neihart Reservior	Wasatch County	Proposed	33.75
09	AZ	Nineteenth Avenue Landfill	Phoenix	Final	54.27
09	CA	Liquid Gold Oil Corp.	Richmond	Final	43.32
09	CA	FMC Corp. (Fresno Plant)	Fresno	Proposed	39.65
09	CA	Sacramento Army Depot	Sacramento	Final	44.46
10	ID	Bunker Hill Mining & Metallurg	Smelterville	Final	54.76
10	OR	Gould, Inc.	Portland	Final	32.12
10	OR	United Chrome Products, Inc.	Corvallis	Final	31.07
10	WA	Harbor Island (Lead)	Seattle	Final	34.60
10	WA	Toftdahl Drums	Brush Prairie	Final	40.22
10	WA	Midway Landfill	Kent	Final	54.27
10	WA	McChord ARB (Wash Rack/Treatment)	Tacoma	Proposed	43.24
10	WA	Nav Undersea Warf Stat (4 Areas)	Keyport	Proposed	33.60

*The governor of a state may grant a waiver to permit listing of a site with a score below 28.5.

APPENDIX G

METHODOLOGICAL DETAILS OF BLOOD-LEAD PREVALENCE PROJECTIONS FROM NHANES II DATA

This Appendix discusses the statistical approaches used for projecting prevalences of blood-lead levels in strata of children and pregnant women in Chapters V and VII. Highlights of the second National Health and Nutrition Examination Survey (NHANES II) methodology and the regression analyses carried out with the NHANES II dataset by J. Schwartz and H. Pitcher of EPA's Office of Policy Analysis are included.

A. THE NHANES II SURVEY

"Because the design of NHANES II is a complex, multistage probability sample, national estimates are derived through a multistage estimation procedure. The procedure has three basic components: (1) inflation by the reciprocal of the probability of selection, (2) adjustment for nonresponse, and (3) poststratification by age, sex, and race. A brief description of each component follows:

- Inflation by the reciprocal of the probability of selection. The probability of selection is the product of the probabilities of selection from each stage of selection in the design -- population sampling unit, segment, household, and sample person.

- Adjustment for nonresponse. The estimates are inflated by a multiplication factor that brings estimates based on examined persons up to a level that would have been achieved if all sample persons had been examined. The nonresponse adjustment factor was calculated by dividing the sum of the reciprocals of the probability of selection for all selected sample persons within each of five income groups (<\$6,000, \$6,000 to \$9,999, \$10,000 to \$14,999, \$15,000 to \$24,999 and ≥\$25,000), three age groups (6 months to 5 years, 6 to 59 years, and 60 to 74 years), four geographic regions, and within or outside SMSAs by the sum of the reciprocals of the probability of selection for examined sample persons in the same income, age, region, and SMSA groups.

- Poststratification by age, sex, and race. The estimates were ratio adjusted within each of 76 age-sex-race cells to independent estimates, provided by the U.S. Bureau of the Census, of the population as of March 1, 1978, the approximate midpoint of the survey. The ratio adjustment used a multiplication factor in which the numerator was the U.S. population and the denominator was the sum of the weights adjusted for nonresponse for examined persons. This ratio estimation process brings the population estimates into agreement with the U.S. Bureau of the Census estimates of the civilian noninstitutionalized U.S. population, and, in general, reduces sampling errors of NHANES II estimates" (Annest and Mahaffey, 1984, p. 41).

Table G-1 presents the arithmetic and geometric means and standard deviations for Pb-B levels of the indicated population segments for the midpoint of the survey, March, 1978.

TABLE G-1. BLOOD-LEAD LEVELS (μg/dl) OF PERSONS 6 MONTHS-74 YEARS, WITH MEANS AND STANDARD DEVIATIONS OF THE MEANS BY SELECTED CHARACTERISTICS: UNITED STATES, 1976-80

Characteristic	Estimated Population in Thousands[a]	Number Examined[b]	Arithmetic		Geometric	
			Mean	Standard Deviation	Mean	Standard Deviation
All persons, 6 months-74 years[c]	203,554	9,936	13.9	6.05	12.8	1.51
All children, 6 months-5 years[c]	16,862	2,376	16.0	6.56	14.9	1.48
White	13,641	1,876	14.9	5.60	14.0	1.44
Black	2,584	420	20.9	8.18	19.6	1.44
All persons, 6-17 years[c]	44,964	1,720	12.5	4.68	11.7	1.45
Men, 18-74 years[c]	67,555	2,798	16.9	6.76	15.8	1.45
Women, 18-74 years[c]	74,173	3,045	11.8	4.64	11.0	1.46

[a] At the midpoint of the survey, March 1, 1978.

[b] With lead determinations from blood specimens drawn by venipuncture.

[c] Includes data for races other than white and black.

Source: Adapted from Annest and Mahaffey (1984), Table X.

B. PROCEDURES USED IN PROJECTING PREVALENCES (ADAPTED FROM U.S. EPA, 1985)

The use of NHANES II data in the models to project the numbers of children and women above various blood-lead levels was a decision by the authors of the report. To estimate the numbers of children above different blood-lead levels in 1984, we relied on both linear and logistic regressions estimated from the NHANES II data. Both regressions were estimated for children aged 6 and under, using only children residing in SMSAs. Independent variables included lead in gasoline and the categorical variables (for race, income, and urbanization) for which separate estimates were given in the tables. These regressions were used to forecast the continued decline in blood-lead levels from 1980, the last year of NHANES II, to 1984. The linear regressions predicted declines in the mean blood-lead level while the logistic regressions modeled changes in the percentage of children above 30 µg/dl. The methodology outlined refers to the original application to gasoline lead-based changes and with the criteria Pb-B levels indicated. Changes in lead content of other sources were not factored in the projections. The criteria Pb-B values of 15, 20, and 25 for projections in this report were handled the same way.

To predict how the number of children above each level would change as the amount of lead in gasoline was reduced, a mechanism was needed to forecast the distribution of blood-lead levels as a function of lead in gasoline. In this analysis, we assumed that the distribution of blood-lead would remain log-normal as gasoline lead levels declined. Then, estimates of the mean and variance of the associated (transformed) normal distribution could be used to determine the percentage of the population above any specific blood-lead level. The estimates of the mean and standard deviation of the underlying normal distribution were derived from logistic regression estimates of the percentage of children with blood-lead levels above 30 µg/dl and linear regression estimates of the mean of the log-normal distribution using the Statistical Analysis System (SAS) procedure, SURREGR.

If the distribution 'X' is normal with mean 'u' and standard deviation 's' (X:N (u,s)), then Y = exp (X) is log-normal with a mean of 'a' and a standard deviation of 'b', where

(1) $a = \exp(u + 1/2\, s^2)$ and

(2) $b = \exp(2u + s^2)(\exp(s^2) - 1)$

Further, if e_g and v_g are the same percentiles of the log-normal and its corresponding normal distribution, respectively, we have

$$(3) \qquad e_g = \exp(u + v_g s)$$

Solving these equations for u and s yielded:

$$(4) \qquad u = \ln(a) - 0.5s^2$$

and

$$(5) \qquad 0 = [\ln(e_g) - \ln(a)] - v_g s + 0.5s^2$$

which had the solution

$$(6) \qquad s = v_g \pm (v_g{}^2 - 2[\ln(e_g) - \ln(a)])^{0.5}$$

Only the smaller root yielded sensible values for u and s. We used the logistic regressions to estimate e_g in equation (3) and the SURREGR regressions to estimate a in equation (1). Using the estimated values for u and s, we determined percentages of the distribution above 10, 15, 20, and 30 μg/dl by looking up the results of $[\ln(10) - u]/s$, etc., in the normal table.

We used a logistic regression equation to estimate the percentage of children over 30 μg/dl to control for problems of multiple sources of exposure. If we had simply used the regressions explaining the mean and assumed a constant standard deviation, we would have predicted that removing lead from gasoline would have resulted in there being no children above 30 μg/dl. This seemed unreasonable because paint and food are known alternate sources of lead, and also are associated with high blood-lead levels. The logistic regressions confirmed that the geometric standard deviation changes as the mean falls.

In previous analyses (U.S. EPA, 1985), this approach was implemented to provide nationwide estimates. Since Congress wanted estimates broken down by more demographic detail in this report, we have run the above procedure separately for each combination of urbanization by race and by income. Obviously, the uncertainty of the estimates for each of these subgroups is much greater than the national estimates.

The same procedure was applied to model the blood-lead distribution for women aged 15 to 45, to produce estimates of fetal exposure to blood-lead levels associated with mental and physical developmental impairment.

Table G-2 provides a tabulation of the child sample sizes for the prevalence modeling of the NHANES II results to the year 1984 contained in Tables V-1, -2, -3, -4, -5, -6, -7, and -8 and corresponding tables in Chapter VII.

TABLE G-2. NHANES II SAMPLE SIZES USED IN MODELING OF BLOOD-LEAD LEVELS[a]

Variable	Number
Race (0.5 - 5 Years)	
White	946
Black	323
Family Income	
<6,000	268
$6,000-14,999	534
≥$15,000	467
SMSA (0.5 - 5 Years)	
Inside Central City	
<1 million	415
≥1 million	262
Outside Central City	
<1 million	347
≥1 million	245

[a] Source: R. S. Murphy, Division of Health Examination Statistics, National Center for Health Statistics.

Generally, the sample sizes are adequate for the estimates of the additive effects assumed in the model. In addition, the model used assumes that no interactions are significant. In EPA's analysis (U.S. EPA, 1985), separate regressions were performed for males, females, blacks, whites, more and less urban, and high and low income; they showed no significant difference in the gasoline lead coefficients among the groups. The sample size was not sufficient to investigate second and third order interactions (e.g., black females or high-income black females); however, the lack of first order interactions makes the assumption of no higher-order interactions plausible. Whether the model is the best one to describe or project blood-lead levels has not been tested.

U.S. GOVERNMENT PRINTING OFFICE: 1988-530-009/84730

603

END

U.S. Dept. of Education

Office of Education
Research and
Improvement (OERI)

ERIC

Date Filmed

March 21,1991